04-25-03

H; Heitmann B L
CORPORATE SOURCE: Department of Medicine TA, Section of Rheumatology, Rigshospitalet, Copenhagen, Denmark.
SOURCE: CLINICAL AND EXPERIMENTAL RHEUMATOLOGY, (1996 May-Jun) 14 (3) 289-93.
Journal code: 8308521. ISSN: 0392-856X.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961204

L5 ANSWER 77 OF 621 MEDLINE
TI Acute thermogenic effects of nicotine and alcohol in healthy male and female smokers.
AB Nicotine intake is associated with lower body weight in both women and men. Despite its energy content, alcohol consumption is also associated with lower body weight in women but not in men. Each drug may reduce weight by acutely increasing thermogenesis. During four sessions, nicotine (20 micrograms/kg per dosing) or placebo was given to male and female smokers (n = 9 each) via measured-dose nasal spray every 30 min for 2 h after consumption of diet tonic water with or without alcohol (0.5 g/kg). Each nicotine/placebo dosing was followed by assessment of energy expenditure by indirect calorimetry. Alcohol alone induced no significant effect in men or women, whereas nicotine alone and combined with alcohol induced a significant thermogenic effect in men but not women. These results are consistent with other research suggesting a reduced thermogenic responsiveness to drugs in women and indicate that nicotine must act via appetite suppression to **reduce body weight** in women. Similarly, these findings do not support the notion that alcohol is inversely related to body weight in women because of excessive acute thermogenesis.
ACCESSION NUMBER: 96397788 MEDLINE
DOCUMENT NUMBER: 96397788 PubMed ID: 8804681
TITLE: Acute thermogenic effects of nicotine and alcohol in healthy male and female smokers.
AUTHOR: Perkins K A; Sexton J E; DiMarco A
CORPORATE SOURCE: Western Psychiatric Institute Clinic, University of Pittsburgh School of Medicine, PA 15213, USA.
CONTRACT NUMBER: DA-04174 (NIDA)
SOURCE: PHYSIOLOGY AND BEHAVIOR, (1996 Jul) 60 (1) 305-9.
Journal code: 0151504. ISSN: 0031-9384.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19990129
Entered Medline: 19961204

L5 ANSWER 78 OF 621 MEDLINE
TI Metformin decreases blood pressure and obesity in OLETF rats via improvement of insulin resistance.
AB To determine whether improvement of insulin resistance decreases blood pressure as well as obesity, metformin (100 mg/kg/d) or vehicle was administered for 20 weeks to 12-week-old male Otsuka Long-Evans Tokushima Fatty (OLETF) rats (n = 10 each), a newly developed animal model of

non-insulin-dependent diabetes mellitus (NIDDM) with mild obesity, hyperinsulinemia, and hypertriglyceridemia. Oral administration of metformin ameliorated glucose intolerance and attenuated the insulin response to glucose loading (2 g/kg, i.p.), as evidenced by a decrease in the area under the curve for glucose and insulin at 24 weeks by 19% and 37%, respectively. At 21 weeks, systolic blood pressure was significantly lower in the metformin group than in controls (130 +/- 1.9 vs. 143 +/- 2.7 mmHg, $p < 0.01$), despite no difference in body weight. Subsequently, blood pressure tended to be slightly but insignificantly lower in the metformin group, and body weight was significantly lower in the metformin group (532 +/- 9.8 vs. 587 +/- 10.3 g at 31 weeks, $p < 0.01$). Metformin treatment also lowered the level of serum triglycerides (9.4 +/- 0.6 vs. 13.2 +/- 0.5 mmol/l, $p < 0.01$) and the plasma norepinephrine concentration (4,222 +/- 373 vs. 7,548 +/- 1,058 pg/ml, $p < 0.01$). These results suggest that metformin-induced improvement of insulin resistance in obese rats with NIDDM may lower blood pressure, as well as decrease sympathetic activity and **reduce body weight**.

ACCESSION NUMBER: 96269257 MEDLINE
DOCUMENT NUMBER: 96269257 PubMed ID: 8829822
TITLE: Metformin decreases blood pressure and obesity in OLETF rats via improvement of insulin resistance.
AUTHOR: Kosegawa I; Katayama S; Kikuchi C; Kashiwabara H; Negishi K; Ishii J; Inukai K; Oka Y
CORPORATE SOURCE: Fourth Department of Medicine, Saitama Medical School, Japan.
SOURCE: HYPERTENSION RESEARCH, (1996 Mar) 19 (1) 37-41. Journal code: 9307690. ISSN: 0916-9636.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961025
Last Updated on STN: 19961025
Entered Medline: 19961017

L5 ANSWER 79 OF 621 MEDLINE
TI Effects of D&C yellow no. 11 ingestion on F344/N rats and B6C3F1 mice.
AB D&C yellow no. 11 (CAS no. 8003-22-3) was administered in the feed at concentrations of 500-50,000 ppm to groups of F344/N rats and B6C3F1 mice of each sex for 13 wk to determine the toxicity. In addition, a perinatal study was conducted to determine the effects of feeding diets containing D&C yellow no. 11 to female rats during reproduction and to their offspring. Although the estimated intake (g/kg) of D&C yellow no. 11 of mice was more than twice that of rats, the results were generally similar for both rats and mice. In both species, D&C yellow no. 11 caused no mortality, but it did **reduce body weight** gain slightly in both sexes of rats exposed to 17,000 and 50,000 ppm. Absolute and relative liver weights were significantly increased in all groups of rats and mice administered D&C yellow no. 11 in the feed. There was minimal to mild degeneration of the periportal hepatocytes in rats at doses of 1700 ppm and higher and in mice at 5000 ppm and above. A dose-related yellow-brown pigment was observed in hepatocytes, Kupffer cells, and biliary epithelium of the liver of both sexes of both species and in the renal tubule epithelium in both sexes of rats. In male rats, all treated groups had increased number and size of hyaline droplets in the renal tubule epithelium of the cortex and outer medulla. To determine if these renal and hepatic lesions were reversible, male rats were administered 5000 ppm dietary D&C yellow no. 11 for 70 d and then examined at 3, 14, and 28 d after the chemical was removed from the diet. Pigment persisted in the kidney and liver for as long as 28 d following removal of D&C yellow no. 11 from the diet, but hepatocellular degeneration and cytoplasmic alteration in the kidney completely resolved by d 3 and 14, respectively. In the perinatal toxicity study, body weight gain in rat

dams given diets containing as much as 50,000 ppm D&C yellow no. 11 for 4 wk before mating to untreated males was similar to that of controls at the time of mating but was lower at parturition and weaning. However, fertility, gestation length, litter size, and pup birth weights were unaffected by treatment. At weaning, there was a significant dose-related decrease in pup body weights from the 5000, 17,000, and 50,000 ppm groups. At 8 wk of age, pups fed the same dosed-feed concentrations as the dams had depressed body weights in the 17,000 and 50,000 ppm treated groups. Microscopic lesions in the liver and kidney of the pups in all dose groups were similar to those described in the 13-wk study. The results of these studies indicate that compound-related effects occurred at all dietary concentrations of D&C yellow no. 11. Liver weights were increased in dosed rats and mice, minimal to mild hepatocellular degeneration was seen in rats receiving dietary concentrations of 1700 ppm and above and in mice at 5000 ppm and above, and there was an increase in the number and size of hyaline droplets in all dosed groups of male rats. Similar compound-related effects were also seen in all dosed rats in the perinatal toxicity study. With the exception of pigment accumulation, the treatment-related kidney and liver lesions in male rats were reversible by 14 d after chemical was withdrawn from the diet.

ACCESSION NUMBER: 96238733 MEDLINE
DOCUMENT NUMBER: 96238733 PubMed ID: 8642626
TITLE: Effects of D&C yellow no. 11 ingestion on F344/N rats and B6C3F1 mice.
AUTHOR: Eastin W C; Elwell M R; Grumbein S; Yuan J H
CORPORATE SOURCE: National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, USA.
SOURCE: JOURNAL OF TOXICOLOGY AND ENVIRONMENTAL HEALTH, (1996 Jun 7) 48 (2) 197-213.
Journal code: 7513622. ISSN: 0098-4108.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960726
Last Updated on STN: 19960726
Entered Medline: 19960717

L5 ANSWER 80 OF 621 MEDLINE
TI Effects of auricular acupuncture stimulation on nonobese, healthy volunteer subjects.
AB Effects of auricular acupuncture stimulation on nonobese healthy volunteers were investigated. Subjects (n = 35) averaged 34.5 years old, and BMI was 25.3 kg/m2. Small (0.15 x 2.0 mm) auricular needles were applied intracutaneously into the bilateral cavum conchae that was identified by having less than 100 k omega resistance. Body weight was measured four times a day and charted by the subjects themselves. Results showed that, in the period 11-2, in which only body weight was measured, without auricular acupuncture stimulations, 57.1% of the subjects reduced their body weight. This indicates that charting body weight themselves might be useful to maintain their weight. In the auricular acupuncture treated period, 19 (70.4%) out of 27 decreased ($p < 0.01$), 5 (18.5%) was increased, and 3 (11.1%) had no change in body weight. In conclusion, the results suggest that success in maintaining weight reduction can be attributed to graphic illustration of one's weight pattern. Bilateral auricular acupuncture stimulation can also **reduce body weight** of healthy non-obese subjects. This is consistent with the suggestion that it might be effective in the treatment of obese patients.

ACCESSION NUMBER: 96231446 MEDLINE
DOCUMENT NUMBER: 96231446 PubMed ID: 8653547
TITLE: Effects of auricular acupuncture stimulation on nonobese, healthy volunteer subjects.
AUTHOR: Shiraishi T; Onoe M; Kageyama T; Sameshima Y; Kojima T;

	Konishi S; Yoshimatsu H; Sakata T
CORPORATE SOURCE:	Department of Neurophysiology, Tokai University School of Medicine, Isehara, Japan.
SOURCE:	OBESITY RESEARCH, (1995 Dec) 3 Suppl 5 667S-673S. Journal code: 9305691. ISSN: 1071-7323.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199607
ENTRY DATE:	Entered STN: 19960808 Last Updated on STN: 19960808 Entered Medline: 19960731

L5 ANSWER 81 OF 621 MEDLINE

TI Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to **reduce body weight** but also to improve insulin sensitivity.

AB OBJECTIVE--To evaluate the effects of walking combined with diet therapy (1,000-1,600 kcal/day) on insulin sensitivity in obese non-insulin-dependent diabetes mellitus (NIDDM) patients. RESEARCH DESIGN AND METHODS--Subjects were divided into two groups: 10 patients were managed by diet alone (group D), and 14 patients were placed in the diet and exercise group (group DE). Group DE was instructed to walk at least 10,000 steps/day on a flat field as monitored by pedometer (19,200 +/- 2,100 steps/day), and group D was told to maintain a normal daily routine (4,500 +/- 290 steps/day). A glucose clamp procedure at an insulin infusion rate of 40 microU.min-2.min-1 was performed before and after the 6- to 8-week training program. Mean serum insulin concentrations ranged from 720 to 790 pmol/l. RESULTS--While body weight (BW) in groups D and DE decreased significantly ($P < 0.01$) during the study, the amount of BW reduction in group DE was greater than that in group D (7.8 +/- 0.8 vs. 4.2 +/- 0.5 kg, $P < 0.01$). After training, glucose infusion rate (GIR) and metabolic clearance rate (MCR) in group D did not significantly increase; however, GIR and MCR increased significantly in group DE, from 17.21 +/- 1.11 to 26.09 +/- 1.11 mumol.kg-1.min-1 ($P < 0.001$) and from 3.0 +/- 0.3 to 5.3 +/- 0.4 ml.kg-1.min-1 ($P < 0.001$), respectively. The analysis of variance showed significant effects of exercise (time x exercise, $P = 0.0005$) for the improvement of MCR. Significant correlations were also observed between delta MCR and average steps per day ($r = 0.7257$, $P < 0.005$) in group DE. CONCLUSIONS--Walking, which can be safely performed and easily incorporated into daily life, can be recommended as an adjunct therapy to diet treatment in obese NIDDM patients, not only for BW reduction, but also for improvement of insulin sensitivity.

ACCESSION NUMBER:	96002685 MEDLINE
DOCUMENT NUMBER:	96002685 PubMed ID: 7555502
TITLE:	Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to **reduce body weight** but also to improve insulin sensitivity.
COMMENT:	Comment in: Diabetes Care. 1999 Oct;22(10):1754-5
AUTHOR:	Yamanouchi K; Shinozaki T; Chikada K; Nishikawa T; Ito K; Shimizu S; Ozawa N; Suzuki Y; Maeno H; Kato K; +
CORPORATE SOURCE:	First Department of Internal Medicine, Aichi Medical University, Japan.
SOURCE:	DIABETES CARE, (1995 Jun) 18 (6) 775-8. Journal code: 7805975. ISSN: 0149-5992.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	(CLINICAL TRIAL) (CONTROLLED CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals

ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 20000407
Entered Medline: 19951121

L5 ANSWER 82 OF 621 MEDLINE
TI Obesity as an adverse prognostic factor for patients receiving adjuvant chemotherapy for breast cancer.
AB OBJECTIVE: To determine whether obesity is an independent prognostic factor among women receiving adjuvant chemotherapy for lymph node-positive breast cancer and to determine how obesity relates to other commonly used prognostic indicators. DESIGN: Retrospective review of the clinical characteristics and clinical course of 735 patients with stages II and III primary breast cancer who were treated using three consecutive postoperative adjuvant chemotherapy protocols. Univariate and multivariate analyses were used to determine the prognostic implications of obesity defined by weight and height tables and body mass index. In addition, we analyzed the relation between obesity and other known prognostic indicators for patients with primary breast cancer. SETTING: A comprehensive cancer center. PATIENTS: 735 patients with lymph node-positive primary breast cancer who were treated using three consecutive fluorouracil-doxorubicin-cyclophosphamide-containing adjuvant chemotherapy protocols and for whom complete data on weight, height, standard prognostic factors, and outcome were available. MAIN OUTCOME MEASUREMENTS: Disease-free and overall survival for the entire group and obese and nonobese subgroups. RESULTS: 24 percent of patients were more than 20% overweight. With a median follow-up of 10.7 years, the estimated 10-year, disease-free rate for patients not more than 20% overweight was 54% (95% CI, 50% to 58%) compared with 40% (CI, 33% to 47%) for remaining patients classified as obese. Although obese patients tended to have somewhat less favorable prognoses based on standard prognostic criteria, a proportional-hazards regression model adjusting for other factors indicated that risk for disease recurrence among obese patients was 1.33 times that of the nonobese population (CI, 1.05 to 1.68). CONCLUSIONS: Obesity is an indicator of poor prognosis for patients with primary breast cancer even after the administration of adjuvant chemotherapy. The effect of dietary interventions to **reduce body weight** on the outcome of breast cancer therapy must be investigated.
ACCESSION NUMBER: 94071322 MEDLINE
DOCUMENT NUMBER: 94071322 PubMed ID: 8250452
TITLE: Obesity as an adverse prognostic factor for patients receiving adjuvant chemotherapy for breast cancer.
AUTHOR: Bastarrachea J; Hortobagyi G N; Smith T L; Kau S W; Buzdar A U
CORPORATE SOURCE: University of Texas M.D. Anderson Cancer Center, Houston.
CONTRACT NUMBER: CA-16672 (NCI)
SOURCE: ANNALS OF INTERNAL MEDICINE, (1994 Jan 1) 120 (1) 18-25. Journal code: 0372351. ISSN: 0003-4819.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199401
ENTRY DATE: Entered STN: 19940201
Last Updated on STN: 19940201
Entered Medline: 19940103

L5 ANSWER 83 OF 621 MEDLINE
TI Insulin sensitivity and lipid levels in obese subjects after slimming diets with different complex and simple carbohydrate content.
AB The ideal hypocaloric diet should **reduce body weight**, decrease fat more than muscle tissue, and ameliorate insulin sensitivity and lipid levels. The aim of this study was to investigate the effect of three hypocaloric diets with different

carbohydrate (CHO) and fat contents on body weight reduction, insulin release and sensitivity, and lipid levels in patients with simple obesity. Twenty-five obese subjects with normal glucose tolerance were randomly allocated to three hypocaloric (800 kcal) diets containing: 60% high complex/high starch and fibre (HC/HSF-CHO) and 20% fat (group 1;11 subjects); 60% high simple/high natural fibre (HS/HNF-CHO) and 20% fat (group 2; 7 subjects); or 20% CHO (L-CHO) and 60% fat (group 3; 7 subjects). The remaining 20% of the diet was protein. In all cases the duration of the diet was 21 days. Before and after the diet, seven subjects from each group underwent a hyperglycemic clamp, and the other four subjects of group 1 underwent a euglycemic-hyperinsulinemic clamp, combined with a glucose turnover study. A similar decrease in body weight, fat-free mass, fat mass, total cholesterol, LDL cholesterol and apo B levels was observed in the three groups. The M/I ratio during hyperglycemic and euglycemic-hyperinsulinemic clamp and the glucose turnover rate during euglycemic-hyperinsulinemic clamp significantly decreased, and FFA levels significantly increased only after the HC/HSF-CHO diet. HDL cholesterol and apo A1 significantly increased only during the HS/HNF-CHO diet.(ABSTRACT TRUNCATED AT 250 WORDS)

ACCESSION NUMBER: 93366522 MEDLINE
DOCUMENT NUMBER: 93366522 PubMed ID: 8395472
TITLE: Insulin sensitivity and lipid levels in obese subjects after slimming diets with different complex and simple carbohydrate content.
AUTHOR: Piatti P M; Pontiroli A E; Saibene A; Santambrogio G; Paroni R; Magni F; Galli-Kienle M; Mistrali S; Monti L D; Pozza G
CORPORATE SOURCE: Istituto Scientifico San Raffaele, Universita di Milano, Italy.
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (1993 Jul) 17 (7) 375-81.
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199309
ENTRY DATE: Entered STN: 19931015
Last Updated on STN: 19980206
Entered Medline: 19930930

L5 ANSWER 84 OF 621 MEDLINE
TI Very low calorie diets and recently developed anti-obesity drugs for treating overweight in non-insulin dependent diabetics.
AB Weight reduction in non-insulin dependent diabetes mellitus (NIDDM) patients improves metabolic control, reduces cardiovascular risk factors, has blood pressure lowering effects and improves the well-being of the patient. This paper describes the role of very low calorie diets (VLCD), exercise, beta-adrenergic drugs and serotoninergic agents in the treatment of overweight in NIDDM. VLCD **reduce body weight** and improve glucose metabolism. Physical exercise programmes in addition to dietary restriction substantially contribute to weight loss and metabolic control in NIDDM. New specific beta-adrenergic agents, exhibiting virtually no beta 1 or beta 2 activity, increase energy expenditure and weight loss probably by enhancement of the basal metabolic rate. The target tissue in humans of this beta-adrenergic effect is as yet unknown. These drugs seem to enhance weight loss when used in combination with (very) low calorie diets compared to dietary restriction alone. Serotoninergic drugs **reduce body weight** by decreasing appetite, in particular for carbohydrates. Furthermore these drugs seem to improve insulin receptor sensitivity.

ACCESSION NUMBER: 93209740 MEDLINE

DOCUMENT NUMBER: 93209740 PubMed ID: 1363597
TITLE: Very low calorie diets and recently developed anti-obesity drugs for treating overweight in non-insulin dependent diabetics.
AUTHOR: Meinders A E; Pijl H
CORPORATE SOURCE: Department of General Internal Medicine, University Hospital, Leiden, The Netherlands.
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (1992 Dec) 16 Suppl 4 S35-9. Ref: 28
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930514
Last Updated on STN: 19970203
Entered Medline: 19930427

L5 ANSWER 85 OF 621 MEDLINE
TI Melatonin inhibits mammary gland development in female mice.
AB The objective of this study was to determine whether melatonin (aMT) influences the postnatal development of the mammary gland parenchyma in female mice from the time of weaning to adulthood. Twenty-one-day-old female BALBc mice were treated with daily subcutaneous injections of aMT (200 micrograms) or diluent, 3 hr before the onset of darkness (photoperiod LD 12:12). At 3, 5, 7, 9, 11, and 13 weeks of age, batches of 20 animals (ten controls and ten aMT-treated) were sacrificed and the second pair of mammary glands were dissected to evaluate their degree of development. Melatonin decreased body weight gain from 2 weeks before until 2 weeks after the onset of puberty. Treatment with aMT also resulted in a lower DNA content and smaller area of the mammary gland from the time of puberty until the end of the study. In aMT-treated mice the phase of highly positive allometric growth began 2 weeks later, but ended at the same time as in controls (11th week of life). Finally, aMT decreased the development of terminal, lateral, and alveolar buds while it increased the number of terminal ducts per gland. We conclude that pharmacological doses of aMT (1) **reduce body weight** gain at the peripuberal age; (2) partially inhibit postnatal mammary gland development by reducing the number of epithelial structures representing sites of growth and increasing that of structures representing the final state of ductal growth in virgin animals; (3) delay the onset of the shorten the phase of rapid mammary growth occurring in early postpuberal age.
ACCESSION NUMBER: 93058330 MEDLINE
DOCUMENT NUMBER: 93058330 PubMed ID: 1432572
TITLE: Melatonin inhibits mammary gland development in female mice.
AUTHOR: Mediavilla M D; San Martin M; Sanchez-Barcelo E J
CORPORATE SOURCE: Department of Physiology and Pharmacology, School of Medicine, University of Cantabria, Spain.
SOURCE: JOURNAL OF PINEAL RESEARCH, (1992 Aug) 13 (1) 13-9.
Journal code: 8504412. ISSN: 0742-3098.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199212
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921209

L5 ANSWER 86 OF 621 MEDLINE

TI Activation of the satiety center by auricular acupuncture point stimulation.

AB Stimulation of the rat inner auricular regions that correspond to the human pylorus, lung, trachea, stomach, esophagus, endocrine, and heart acupuncture points evoked potentials in the hypothalamic ventromedial nucleus (HVM), the satiety center. Needle implantation into any of these points reduced the body weight to its initial 290 g after the rat had gained about 410 g in 20 days, and significantly reduced initial 450-g body weights (p less than 0.01, Student's t test) in 14 days. Stimulation of other acupuncture points did not evoke HVM potentials and did not **reduce body weight**. After the HVM was lesioned, body weight increased and acupuncture point needling had no effect on body weight. Needling of the auricular acupuncture points evoked no potentials in the lateral hypothalamus (LHA), the feeding center, and had almost no influence on weight reduction induced by LHA lesion.

ACCESSION NUMBER: 92404933 MEDLINE
DOCUMENT NUMBER: 92404933 PubMed ID: 1525671
TITLE: Activation of the satiety center by auricular acupuncture point stimulation.
AUTHOR: Asamoto S; Takeshige C
CORPORATE SOURCE: Department of Physiology, Showa University School of Medicine, Tokyo, Japan.
SOURCE: BRAIN RESEARCH BULLETIN, (1992 Aug) 29 (2) 157-64. Journal code: 7605818. ISSN: 0361-9230.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199210
ENTRY DATE: Entered STN: 19921106
Last Updated on STN: 19921106
Entered Medline: 19921022

L5 ANSWER 87 OF 621 MEDLINE

TI Long-term exercise training and retirement in genetically obese rats: effects on food intake, feeding efficiency and carcass composition.

AB Short-term physical exercise (EX) can **reduce body weight** and fat gain in obese humans and animals. However, the beneficial effects of physical exercise are not long-lasting. In this study, the effects of long-term physical exercise and retirement from exercise (R) on body weight, body composition and fat distribution were examined in genetically obese (OB) and lean (LE) female rats. Fifty OB and 45 LE rats, four weeks old, were divided into EX (swimming, 2h/day, 5 days/week) or sedentary (SD) groups. At the end of the 28th week of treatment, EX groups were further divided into continued EX or R groups for another 11-12 weeks. It was found that at the end of the 28th week EX had reduced the rate of weight gain in OB and LE rats. Percentage body fat was only reduced in OBEX rats and this was achieved by a significant reduction of subcutaneous fat mass. At the end of the 40th week, EX had further reduced the weight gain, fat mass and body fat percentage in OBEX rats while only body fat percentage was reduced in the LEEX group. Retirement from exercise reversed these phenomena. Thus there were no differences between OBSD and OBEX-R rats in body weight, fat mass and percentage body fat. However, the OBEX-R group had a significantly higher amount of internal fat than the other two OB groups. Therefore, exercise, even long-term to cover the entire fat cell proliferation period, still only exerted temporary beneficial effects in OB rats. After retirement, the beneficial effects all disappeared rapidly.(ABSTRACT TRUNCATED AT 250 WORDS)

ACCESSION NUMBER: 92363688 MEDLINE
DOCUMENT NUMBER: 92363688 PubMed ID: 1323548
TITLE: Long-term exercise training and retirement in genetically

obese rats: effects on food intake, feeding efficiency and carcass composition.
AUTHOR: Jen K L; Almario R; Ilagan J; Zhong S; Archer P; Lin P K
CORPORATE SOURCE: Department of Nutrition and Food Science, Wayne State University, Detroit, Michigan 48202.
CONTRACT NUMBER: DK40046 (NIDDK)
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (1992 Jul) 16 (7) 519-27.
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199209
ENTRY DATE: Entered STN: 19920925
Last Updated on STN: 19920925
Entered Medline: 19920911

L5 ANSWER 88 OF 621 MEDLINE
TI Inhibition of growth in chickens by testosterone, 5 alpha-dihydrotestosterone, and 19-nortestosterone.
AB The growth response of poultry to androgens is ambiguous, with both increases and decreases being reported. This may reflect the use of pharmacological doses. The present study examined the effect of physiological concentrations of androgens on growth of intact male, intact female, and castrated chickens. Physiological concentrations of androgen were attained by subcutaneous silastic implants. In mammals, androgens have both androgenic effects on the reproductive organs and anabolic growth-promoting effects on body and muscle growth. Some androgens, for instance 5 alpha-dihydrotestosterone (5 alpha-DHT) have high androgenic activity (5 alpha-DHT greater than testosterone) but others, e.g., 19-nortestosterone, have high anabolic activities (19-nortestosterone greater than testosterone greater than 5 alpha-DHT). The relative effects of testosterone, 5 alpha-DHT, and 19-nortestosterone on growth were compared in chickens. In young, intact male and female chicks, growth was suppressed by 1.0-cm silastic implants of testosterone and 5 alpha-DHT (5 alpha-DHT greater than testosterone). Castrated chicks were implanted with implants of various sizes (.3, 1.0, and 3.0 cm) containing testosterone, 5 alpha-DHT, or 19-nortestosterone. The androgens inhibited body weight gain: 19-nortestosterone reducing body weight at all three doses, 5 alpha-DHT reducing body weight at the intermediate and high doses, and testosterone tending to **reduce body weight** only at the high dose. Testosterone (3.0 cm), 5 alpha-DHT (all doses), and 19-nortestosterone (all doses) reduces skeletal growth, as indicated by shank-toe length. In contrast to their growth-suppressing effect, all three steroids exerted an androgenic effect; stimulating comb and wattle development (19-nortestosterone greater than 5 alpha-DHT greater than testosterone). It is concluded that androgens are androgenic but are not anabolic in chickens.
ACCESSION NUMBER: 92187495 MEDLINE
DOCUMENT NUMBER: 92187495 PubMed ID: 1546048
TITLE: Inhibition of growth in chickens by testosterone, 5 alpha-dihydrotestosterone, and 19-nortestosterone.
AUTHOR: Fennell M J; Scanes C G
CORPORATE SOURCE: Department of Animal Sciences, Rutgers-The State University, New Brunswick, New Jersey 08903.
SOURCE: POULTRY SCIENCE, (1992 Feb) 71 (2) 357-66.
Journal code: 0401150. ISSN: 0032-5791.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199204
ENTRY DATE: Entered STN: 19920424

Last Updated on STN: 19920424
Entered Medline: 19920410

L5 ANSWER 89 OF 621 MEDLINE
TI Overview of adrenergic anorectic agents.
AB Adrenergic anorexic agents of the amphetamine class suppress appetite and **reduce body weight** via activation of beta-adrenergic and/or dopaminergic receptors within the perifornical hypothalamus (PFH). Although phenylpropanolamine (PPA) is often considered to be a member of the amphetamine class of anorexiants, this drug is an atypical adrenergic anorexiant. Unlike amphetamine, microinjection of PPA into the PFH does not suppress feeding. Moreover, PPA anorexia is not reversed by the dopamine antagonist haloperidol. The anorexic action of PPA may result, in part, from its interaction with alpha 1-adrenergic receptors within the paraventricular medial hypothalamus (PVN). This hypothesis is supported by prior research, which documents that PPA is a direct-acting agonist predominantly at alpha 1 adrenoceptors, that microinjections into the PVN of the alpha 1-adrenoceptor agonists PPA and l-phenylephrine suppress feeding, and that injections of alpha 1-adrenoceptor antagonists within the PVN enhance feeding behavior.

ACCESSION NUMBER: 92101947 MEDLINE
DOCUMENT NUMBER: 92101947 PubMed ID: 1309478
TITLE: Overview of adrenergic anorectic agents.
AUTHOR: Wellman P J
CORPORATE SOURCE: Department of Psychology, Texas A&M University, College Station, TX 77843-4235.
SOURCE: AMERICAN JOURNAL OF CLINICAL NUTRITION, (1992 Jan) 55 (1 Suppl) 193S-198S. Ref: 31
Journal code: 0376027. ISSN: 0002-9165.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920223
Last Updated on STN: 19970203
Entered Medline: 19920203

L5 ANSWER 90 OF 621 MEDLINE
TI [Evaluation of a weight reduction program: slender without diets]. Evaluation des Gewichtsreduktionsverfahrens: Schlank ohne Diat.
AB "Schlank ohne Diat" ("Weight-Reduction Without Diet") is a strategy to normalize body weight, by influencing multiple factors that appear to influence and promote obesity. The base line of therapy is the modification of nutritional habits. Self-control, especially monitoring and recording of calorie intake and the loss of energy by physical activities is the key that trains every client to change his nutritional habits and helps to **reduce body weight** and keep normal body weight stable. In a retrospective study, including 134 persons, 84 clients (62,69%) were able to **reduce body weight**, 9 clients (6,72%) reached starting point of weight and in 30,60% (41 clients) during the participation in this methods an increase of body weight was seen. On an average 120 clients achieved a weight reduction of 5.98 kg during the participation in this method. The loss of weight ranged from 1 to 31 kg per person.

ACCESSION NUMBER: 91377269 MEDLINE
DOCUMENT NUMBER: 91377269 PubMed ID: 1897283
TITLE: [Evaluation of a weight reduction program: slender without diets].
Evaluation des Gewichtsreduktionsverfahrens: Schlank ohne Diat.

AUTHOR: Kiefer I; Schoberberger R; Kunze M
CORPORATE SOURCE: Institut fur Sozialmedizin, Universitat Wien.
SOURCE: ZEITSCHRIFT FUR DIE GESAMTE INNERE MEDIZIN UND IHRE GRENZGEBIETE, (1991 May) 46 (7) 255-9.
Journal code: 21730470R. ISSN: 0044-2542.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 19911108
Last Updated on STN: 19911108
Entered Medline: 19911024

L5 ANSWER 91 OF 621 MEDLINE

TI Anticoccidial activity of 8-aminoquinolines, pamaquine, primaquine and several molecular complexes and salts of pamaquine, against Eimeria tenella, E. necatrix, E. acervulina, E. maxima, and E. brunetti in battery experiments.

AB Pamaquine and primaquine, the well known antimalarial 8-aminoquinolines, have not been reported for their anticoccidial activity. A series of battery experiments was conducted to investigate their activity against a laboratory strain of Eimeria tenella, E. necatrix, E. acervulina, E. maxima, or E. brunetti and revealed that both drugs were effective against E. tenella and E. necatrix, but not against the other three species. Pamaquine suppressed the symptoms of E. tenella induced coccidiosis at concentrations above 125 ppm in feed and primaquine controlled the clinical signs as well at levels above 31.2 ppm. The activity against E. necatrix was observed with pamaquine at 250 ppm and with primaquine at levels above 125 ppm. Pamaquine showed a tendency apparently to **reduce body weight** gain at 125-500 ppm, whereas primaquine showed the same tendency at 500 ppm. In a concomitantly conducted experiment, this adverse effect of pamaquine was averted in its molecular complexes with benzophenone, nitropyrazole, dinitrobenzoic acids and quinoline, and in its salts of sulfate or zinc chloride, and yet these compounds retained the same anticoccidial activity as of pamaquine. This suggests that these compounds had the broadened safety margin. Judging from their susceptibility to these compounds. E. tenella and E. necatrix will have similar metabolic functions to those of blood cell parasitizing protozoa like plasmodia and prioplasma, which are easily suppressed by this class of compound.

ACCESSION NUMBER: 91322214 MEDLINE
DOCUMENT NUMBER: 91322214 PubMed ID: 1830767
TITLE: Anticoccidial activity of 8-aminoquinolines, pamaquine, primaquine and several molecular complexes and salts of pamaquine, against Eimeria tenella, E. necatrix, E. acervulina, E. maxima, and E. brunetti in battery experiments.
AUTHOR: Matsuno T; Hariguchi F; Okamoto T
CORPORATE SOURCE: Animal Health Research Laboratories, Agro Division, Takeda Chemical Industries, Ltd., Osaka, Japan.
SOURCE: JOURNAL OF VETERINARY MEDICAL SCIENCE, (1991 Feb) 53 (1) 13-7.
Journal code: 9105360. ISSN: 0916-7250.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199109
ENTRY DATE: Entered STN: 19910929
Last Updated on STN: 19910929
Entered Medline: 19910912

L5 ANSWER 92 OF 621 MEDLINE

TI [Screening for obesity in a schoolchildren population of the 20th zone of Milan and a nutritional education intervention].
Screening dell'obesita nella popolazione scolastica della zona 20 di Milano ed intervento di educazione alimentare.

AB Our study was performed in 1986-'87 and 1987-'88 school years on 12.354 three to eighteen years old students (the whole scholastic population of zone 20 of Milan) in order to apply dietary education on obese subjects. Mean prevalence of obesity was 13.4% with elevated percentages in 11 to 13 years old students (17.9%), with respect to primary (14.1%), high school (12.4%) and nursery school (4.7%). The 36% of obese subjects (more than 50% of adolescents) had already tempted to **reduce body weight**. Intervention reduced % weight excess (from 33.6 +/- 0.5% to, 28.8 +/- 0.5% after 12 months, p less than 0.001); 67% of obese subjects lost weight and body weight returned within normal limits in 31% of subjects. An educational dietetic strategy may be successful in childhood obesity.

ACCESSION NUMBER: 91249974 MEDLINE
DOCUMENT NUMBER: 91249974 PubMed ID: 2151321
TITLE: [Screening for obesity in a schoolchildren population of the 20th zone of Milan and a nutritional education intervention].
Screening dell'obesita nella popolazione scolastica della zona 20 di Milano ed intervento di educazione alimentare.
AUTHOR: Ceratti F; Garavaglia M; Piatti L; Brambilla P; Rondanini G F; Bolla P; Ghisalberti C; Chiumello G
CORPORATE SOURCE: USL, Servizio di Medicina Scolastica, Milano.
SOURCE: EPIDEMIOLOGIA E PREVENZIONE, (1990 Dec) 12 (45) 1-6.
Journal code: 8902507. ISSN: 1120-9763.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 19910728
Last Updated on STN: 19910728
Entered Medline: 19910711

L5 ANSWER 93 OF 621 MEDLINE

TI Role for brain corticotropin-releasing factor in the weight-reducing effects of chronic fenfluramine treatment in rats.

AB Fenfluramine is an amphetamine derivative which is used as a weight-reducing agent in the treatment of obesity. It has been postulated that fenfluramine affects brain serotonin (5HT) neurons resulting in decreased food intake and altered autonomic outflow which, in turn, increases metabolism. CRF decreases food intake and, in addition, has been demonstrated to **reduce body weight** in genetically obese rats through selective activation of sympathetic and inhibition of parasympathetic outflows. Because 5HT is a potent CRF secretagogue, we tested the hypothesis that the weight-reducing effects of fenfluramine administration may be mediated, in part, through altered CRF secretion. Chronic fenfluramine treatment (1-24 mg/kg sc, twice daily, 4 days) resulted in a dose-dependent decrease in hypothalamic CRF concentration at 30 min after the final drug injection and was accompanied by a significant reciprocal increase in plasma corticosterone concentration. These data suggest that the decrease in hypothalamic CRF was a consequence of increased CRF secretion. These changes in hypothalamic CRF and plasma corticosterone correlated with brain fenfluramine levels. In contrast, high dose fenfluramine treatment significantly increased hippocampus, midbrain, and spinal cord CRF concentrations whereas levels in cerebral cortex, caudate putamen, thalamus, pons/medulla, and cerebellum were unaffected. There was no effect of this fenfluramine treatment protocol on regional brain TRH or neurotensin concentrations. In keeping with the well known development of tolerance to the weight-reducing effects of fenfluramine, chronic

fenfluramine treatment resulted in lesser increases in corticosterone secretion than after acute treatment. Whereas weight loss observed after chronic fenfluramine treatment was associated with stimulation of hypothalamic-pituitary-adrenocortical hormone secretion, the weight-recovery phase after cessation of drug treatment was associated with decreased levels of plasma corticosterone. These data, demonstrating fenfluramine-induced alterations in brain CRF and plasma corticosterone, suggest that CRF may represent an important endogenous transmitter which mediates the weight-reducing effects of the drug.

ACCESSION NUMBER: 91243671 MEDLINE
DOCUMENT NUMBER: 91243671 PubMed ID: 1645265
TITLE: Role for brain corticotropin-releasing factor in the weight-reducing effects of chronic fenfluramine treatment in rats.
AUTHOR: Appel N M; Owens M J; Culp S; Zaczek R; Contrera J F; Bissette G; Nemeroff C B; De Souza E B
CORPORATE SOURCE: Neurobiology Laboratory, NIDA Addiction Research Center, Baltimore, Maryland 21224.
CONTRACT NUMBER: MH-39415 (NIMH)
MH-42088 (NIMH)
SOURCE: ENDOCRINOLOGY, (1991 Jun) 128 (6) 3237-46.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199107
ENTRY DATE: Entered STN: 19910719
Last Updated on STN: 19910719
Entered Medline: 19910702

L5 ANSWER 94 OF 621 MEDLINE
TI Insulin infusion stimulates daily food intake and body weight gain in diabetic rats.
AB Current theories state that physiological levels of insulin inhibit daily food intake and **reduce body weight**. To test whether insulin induces satiety, systematically increasing doses of insulin from 2.0 to 5.0 U/day were infused intravenously into streptozotocin-induced diabetic rats. Food intake increased significantly from 70.0 +/- 1.4 kcal/day during the saline baseline up to 102.2 +/- 1.9 kcal/day in the 3.5 U/day insulin infusion and then stabilized at 95.9 +/- 0.5 kcal/day for the remaining doses (p less than 0.05). Retained energy values (kcal of food intake minus kcal of urinary glucose loss) also increased from 69.9 +/- 1.4 kcal/day to stabilize at 95 kcal/day (p less than 0.001). Food intake and retained energy of normal controls remained unchanged at 75.4 +/- 1.6 kcal/day for the duration of the study. With elevated food intake and retained energy values after the 3.5 U/day insulin dose, the diabetic rats gained more weight than the normal controls (p less than 0.01). Contrary to expectations, increasing the amount of insulin infused through the physiological range results in a 40% increase in daily food intake and a rapid gain in body weight.

ACCESSION NUMBER: 91204802 MEDLINE
DOCUMENT NUMBER: 91204802 PubMed ID: 2087522
TITLE: Insulin infusion stimulates daily food intake and body weight gain in diabetic rats.
AUTHOR: Willing A E; Walls E K; Koopmans H S
CORPORATE SOURCE: Department of Medical Physiology, University of Calgary, Alberta, Canada.
CONTRACT NUMBER: DK 40217 (NIDDK)
SOURCE: PHYSIOLOGY AND BEHAVIOR, (1990 Dec) 48 (6) 893-8.
Journal code: 0151504. ISSN: 0031-9384.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 19910607
Last Updated on STN: 19910607
Entered Medline: 19910523

L5 ANSWER 95 OF 621 MEDLINE
TI [Evaluation of the weight reduction program "Reducing without Diet"].
Evaluation des Gewichtsreduktionsverfahrens "Schlank ohne Diat".
AB "Schlank ohne Diat" ("Weight-Reduction Without Diet") is a strategy to normalize body weight, by influencing multiple factors that appear to influence and promote obesity. The base line of therapy is the modification of nutritional habits. Self-control, especially monitoring and recording of caloric intake and the loss of energy by physical activities is the key that trains every client to change his nutritional habits and helps to **reduce body weight** and keep normal body weight stable. In a retrospective study, including 134 persons, 84 clients (62.69%) were able to **reduce body weight**, 9 clients (6.72%) reached starting point of weight and in 30, 60% (41 clients) during the participation in this methods an increase of body weight was seen. On an average 120 clients achieved a weight reduction of 5.98 kg during the participation in this methode. The loss of weight ranged from 1 to 31 kg per person.
ACCESSION NUMBER: 91133683 MEDLINE
DOCUMENT NUMBER: 91133683 PubMed ID: 2149446
TITLE: [Evaluation of the weight reduction program "Reducing without Diet"].
Evaluation des Gewichtsreduktionsverfahrens "Schlank ohne Diat".
AUTHOR: Kiefer I; Schoberberger R; Kunze M
CORPORATE SOURCE: Institut fur Sozialmedizin der Universitat Wien.
SOURCE: OFFENTLICHE GESUNDHEITSWESEN, (1990 Dec) 52 (12) 703-7.
Journal code: 0107170. ISSN: 0029-8573.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199103
ENTRY DATE: Entered STN: 19910405
Last Updated on STN: 19970203
Entered Medline: 19910321

L5 ANSWER 96 OF 621 MEDLINE
TI Hyperlipidemia treated with xiaobu jianfei pian.
AB A total of 51 cases with hyperlipidemia, who were defined deficiency symptom-complex complicated by symptoms of excessiveness in TCM were studied clinically. The patients were divided into two groups at random. One group was treated with Xiaobu Jianfei Pian (XJP) as treated group, another with Fangfeng Tongsheng San as a control. It was found that XJP was able to lower total serum cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and apolipoprotein (apo) B significantly (P less than 0.001, 0.001, 0.001) while it had markedly improved clinical symptoms. It was also observed that XJP had good effects on the ratios of apoA1/B and TC/HDL-C, and was able to **reduce body weight** index. All of these were better than those of the control group statistically. These evidences indicate that XJP possesses clinical therapeutic effects on both lipid-lowering and lipid-adjusting, which suggest that XJP may be an effective anti-hyperlipidemia medicine.
ACCESSION NUMBER: 91098753 MEDLINE
DOCUMENT NUMBER: 91098753 PubMed ID: 2268940
TITLE: Hyperlipidemia treated with xiaobu jianfei pian.
AUTHOR: Qiu W S; Chen K J; Li C S
CORPORATE SOURCE: Xiyuan Hospital, China Academy of TCM, Beijing.
SOURCE: CHUNG HSI I CHIEH HO TSA CHIH CHINESE JOURNAL OF MODERN

DEVELOPMENTS IN TRADITIONAL MEDICINE, (1990 Sep) 10 (9) 532-4, 516.
Journal code: 8207427. ISSN: 0254-9034.
PUB. COUNTRY: China
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199102
ENTRY DATE: Entered STN: 19910329
Last Updated on STN: 19950206
Entered Medline: 19910220

L5 ANSWER 97 OF 621 MEDLINE

TI Does emotional eating interfere with success in attempts at weight control?.

AB Questionnaire responses from a convenience sample were used to test for hypothesized relationships between changes over time in individuals' reported frequency of emotional eating and estimates of their success in attempts to **reduce body weight** over periods of at least 1 year. Respondents were 187 English adults, whose distribution of estimated Body Mass Indices (BMI) approximately that of the general population. Initial BMI was significantly (p less than 0.001) positively associated with reported frequency of emotional eating. Moreover, respondents indicating initially relatively high levels of emotional eating who reported a reduction in that level were found to lose significantly (p less than 0.01) more reported weight and to be significantly (p less than 0.05) more successful at approaching target weight over the period of the study than respondents who continued to report high levels of emotional eating. Similarly, respondents who reported an increase from initially relatively low levels of emotional eating, while not losing significantly less reported weight, were significantly (p less than 0.05) less successful at approaching target weight than those respondents who continued to report low levels of emotional eating.

ACCESSION NUMBER: 91097204 MEDLINE
DOCUMENT NUMBER: 91097204 PubMed ID: 2268140
TITLE: Does emotional eating interfere with success in attempts at weight control?.
AUTHOR: Blair A J; Lewis V J; Booth D A
CORPORATE SOURCE: School of Psychology, University of Birmingham, U.K.
SOURCE: APPETITE, (1990 Oct) 15 (2) 151-7.
Journal code: 8006808. ISSN: 0195-6663.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199102
ENTRY DATE: Entered STN: 19910322
Last Updated on STN: 19910322
Entered Medline: 19910212

L5 ANSWER 98 OF 621 MEDLINE

TI Involvement of corticotropin-releasing factor in the anorexia induced by exercise.

AB The role of corticotropin-releasing factor (CRF) in the sex-dependent anorexia induced by exercise was investigated in male Wistar rats. Each rat was implanted with a permanent guide cannula that was stereotaxically positioned close to the right lateral ventricle of the brain. During the recovery period, which lasted 10 days, rats were accustomed to eat three meals per day. The onset of each meal occurred every 8 hours. During a meal, rats were allowed free access to a pelleted stock diet for one hour. Ten days after the surgery, rats were injected in the right lateral

ventricle of the brain with either saline or 100 micrograms of alpha-helical CRF (9-41), a selective CRF antagonist. Fifteen minutes after the injections, half of the rats were forced to exercise while the others were allowed to rest. The exercise consisted of a moderately intense period of running on a motor-driven treadmill during 40 minutes. Immediately after the period of exercise, animals were offered food, and the amount eaten during the meal period was carefully measured. The results indicate that the exercised rats ate less food than resting animals when saline was infused prior to subjecting the animals to running. In addition, in saline-treated animals, growth of exercised rats was slower than that of resting rats during the day following exercise. Contrastingly, in rats infused with alpha-helical CRF (9-41) exercise exerted no effect on food intake, neither did it **reduce body weight** gain of the rats. The present results suggest that CRF plays a major role in the anorexia caused by exercise in male rats.

ACCESSION NUMBER: 91003453 MEDLINE
DOCUMENT NUMBER: 91003453 PubMed ID: 2207704
TITLE: Involvement of corticotropin-releasing factor in the anorexia induced by exercise.
AUTHOR: Rivest S; Richard D
CORPORATE SOURCE: Departement de Physiologie, Faculte de Medecine, Universite Laval, Quebec, Canada.
SOURCE: BRAIN RESEARCH BULLETIN, (1990 Jul) 25 (1) 169-72. Journal code: 7605818. ISSN: 0361-9230.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199011
ENTRY DATE: Entered STN: 19910117
Last Updated on STN: 19910117
Entered Medline: 19901114

L5 ANSWER 99 OF 621 MEDLINE
TI Effects of intermittent feeding upon body weight and lifespan in inbred mice: interaction of genotype and age.
AB Beginning at either 1.5, 6 or 10 months of age, male mice from the A/J and C57BL/6J strains and their F1 hybrid, B6AF1/J were fed a diet (4.2 kcal/g) either ad libitum every day or in a restricted fashion by ad libitum feeding every other day. Relative to estimates for ad libitum controls, the body weights of the intermittently-fed restricted C57BL/6J and hybrid mice were reduced and mean and maximum life span were incremented when the every-other-day regimen was initiated at 1.5 or 6 months of age. When every-other-day feeding was introduced at 10 months of age, again both these genotypes lost body weight relative to controls; however, mean life span was not significantly affected although maximum life span was increased. Among A/J mice, intermittent feeding did not **reduce body weight** relative to ad libitum controls when introduced at 1.5 or 10 months of age; however, this treatment did increase mean and maximum life span when begun at 1.5 months, while it decreased mean and maximum life span when begun at 10 months. When restricted feeding was introduced to this genotype at 6 months of age, body weight reduction compared to control values was apparent at some ages, but the treatment had no significant effects on mean or maximum life span. These results illustrate that the effects of particular regimens of dietary restriction on body weight and life span are greatly dependent upon the genotype and age of initiation. Moreover, when examining the relationship of body weight to life span both between and within the various groups, it was clear that the complexity of this relationship made it difficult to predict that lower body weight would induce life span increment.

ACCESSION NUMBER: 90384210 MEDLINE
DOCUMENT NUMBER: 90384210 PubMed ID: 2402168

TITLE: Effects of intermittent feeding upon body weight and lifespan in inbred mice: interaction of genotype and age.
AUTHOR: Goodrick C L; Ingram D K; Reynolds M A; Freeman J R; Cider N
CORPORATE SOURCE: Nathan W. Shock Laboratories, Gerontology Research Center, Baltimore, MD 21224.
SOURCE: MECHANISMS OF AGEING AND DEVELOPMENT, (1990 Jul) 55 (1) 69-87.
Journal code: 0347227. ISSN: 0047-6374.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 19901122
Last Updated on STN: 19901122
Entered Medline: 19901024

L5 ANSWER 100 OF 621 MEDLINE
TI [Fasting--wrong in obesity?].
Fasta--fel vid fetma?.
AB Fasting has been advocated as an effective way to **reduce body weight**. However, few data support any long-term effect of this therapy. On the contrary, evidence is accumulating that the repeated weight loss and concomitant weight gain, typical of fasting in many individuals, will lead to a subsequently higher body weight. Each weight cycle seems to increase the risk of a higher waist/hip ratio, greater metabolic efficiency and a food preference towards fat. All these trends result in further problems associated with weight loss and a vicious circle is established.
ACCESSION NUMBER: 90310664 MEDLINE
DOCUMENT NUMBER: 90310664 PubMed ID: 2367191
TITLE: [Fasting--wrong in obesity?].
Fasta--fel vid fetma?.
AUTHOR: Rossner S
CORPORATE SOURCE: Obesitasenheten, Medicinska kliniken, Karolinska sjukhuset, Stockholm.
SOURCE: NORDISK MEDICIN, (1990) 105 (6-7) 190-1.
Journal code: 0401001. ISSN: 0029-1420.
PUB. COUNTRY: Sweden
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Swedish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199008
ENTRY DATE: Entered STN: 19900921
Last Updated on STN: 19900921
Entered Medline: 19900813

L5 ANSWER 101 OF 621 MEDLINE
TI Effects of chronic ethanol diet on pituitary-testicular function of the rat.
AB We studied the effects of a 6% ethanol liquid diet administered for 5 wk on the pituitary-gonadal function of adult male rats. Because ethanol is known to **reduce body weight**, we used sucrose-fed animals as controls. No significant differences in body, testis, or prostate weights were found between the rats exposed to ethanol and their sucrose-fed controls at the end of the 5-week treatment. Seminal vesicle weights decreased significantly (p less than 0.05) in the ethanol-treated group. Serum and testicular testosterone concentrations were significantly reduced in the ethanol-treated group, to 43.6% and 48.3% of levels in the sucrose-fed controls, respectively (p less than 0.05). Serum luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels of the ethanol-treated rats were 37.9% and 41.3%, respectively, of those of the sucrose-fed controls (p less than

0.01-0.05). The pituitary levels of these hormones were similar to those of controls, but the ratios of pituitary LH and FSH to their serum levels were clearly increased after ethanol exposure, to 492% and 206.1%, respectively (p less than 0.05). In contrast, pituitary prolactin (PRL) of the ethanol-treated rats was decreased to 40.2% (p less than 0.01) of sucrose-fed controls. Testicular content of LH receptors was significantly reduced (to 77.0% of controls; p less than 0.01), but content of FSH receptors was slightly increased by the ethanol diet (to 121.5% of sucrose-fed controls; p less than 0.05). No ethanol-associated changes were apparent in testicular PRL and gonadotropin-releasing hormone (GnRH) receptors or in pituitary GnRH receptors.(ABSTRACT TRUNCATED AT 250 WORDS)

ACCESSION NUMBER: 90181578 MEDLINE
DOCUMENT NUMBER: 90181578 PubMed ID: 2155675
TITLE: Effects of chronic ethanol diet on pituitary-testicular function of the rat.
AUTHOR: Salonen I; Huhtaniemi I
CORPORATE SOURCE: Department of Anatomy, University of Turku, Finland.
SOURCE: BIOLOGY OF REPRODUCTION, (1990 Jan) 42 (1) 55-62. Journal code: 0207224. ISSN: 0006-3363.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199004
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19980206
Entered Medline: 19900423

L5 ANSWER 102 OF 621 MEDLINE
TI Effects of water temperature and flavoring on voluntary dehydration in men.
AB Effects of water temperature and flavoring on fluid consumption and body weight losses were studied in fourteen unacclimatized men (21-33 years) during 6 hr of treadmill exercise (4.8 km.hr-1, 5% grade for 30 min.hr-1) in a hot environment. Subjects consumed each of four beverages (15 degrees C water, 40 degrees C water, 15 degrees C flavored water, and 40 degrees C flavored water) on four nonconsecutive days. We identified two groups of individuals by body weight (BW) loss during the cool water trial: drinkers (D) who lost less than 2% initial BW (0.80 +/- 0.15%) and reluctant drinkers (RD) who lost more than 2% (2.53 +/- 0.12%). Although sweat losses were not different between the two groups, D consumed 31% more cool water than RD and experienced 68% less BW loss. Compared to the warm water trial, 6 hr consumption of cool water was significantly increased in both D (59%) and RD (141%) and BW loss was dramatically reduced in both groups. Flavoring significantly enhanced warm water consumption and reduced BW loss in RD only. Reduced consumption of warm water increased rectal temperature, heart rate and plasma osmolality in both groups. The results of this study indicate that either flavoring or cooling warm water will enhance fluid intake and **reduce body weight** deficits in men reluctant to drink.

ACCESSION NUMBER: 89331724 MEDLINE
DOCUMENT NUMBER: 89331724 PubMed ID: 2756057
TITLE: Effects of water temperature and flavoring on voluntary dehydration in men.
AUTHOR: Szlyk P C; Sils I V; Francesconi R P; Hubbard R W; Armstrong L E
CORPORATE SOURCE: U.S. Army Research Institute of Environmental Medicine, Heat Research Division, Natick, MA 01760-5007.
SOURCE: PHYSIOLOGY AND BEHAVIOR, (1989 Mar) 45 (3) 639-47. Journal code: 0151504. ISSN: 0031-9384.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198909
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19900309
Entered Medline: 19890905

L5 ANSWER 103 OF 621 MEDLINE
TI Phenobarbital during pregnancy in mouse and man.
AB Phenobarbital appears to produce similar behavioral effects on mice and humans with excitation at low and sedation or depression at higher doses. If plasma concentrations of phenobarbital reflect levels in other tissue, then brain concentrations producing excitation (near 10 micrograms/g) and depression (near 20 micrograms/g) are not substantially different for the two species. The doses needed to produce these levels are much higher in mice than man. Plasma concentrations of phenobarbital decline during pregnancy in humans. Whether this decline is accompanied by increased seizure frequency has not been confirmed empirically and whether pregnancy influences the frequency of seizures at all is controversial. The reduction in phenobarbital levels in plasma or serum during pregnancy has been confirmed in rodents. Two of these studies however reported no difference brain concentrations of the drug during pregnancy. One study indicated increased potency of the drug, however this was not confirmed by the other two reports. The effects of phenobarbital on the progression of pregnancy and on offspring is not well defined in humans partly because the disease and the treatment effects are confounded. There are a few studies however which suggest that the effects might be drug specific. Animal studies in this area differ substantially from humans in design making any comparison tentative. The effects of the drug on pregnancy and neonates in rodents depends on the method of administering the drug and dose. Drug administration via the diet can provide high blood levels in the dams and causes lowered birthweight as well as several anatomical and hormonal abnormalities in offspring. This procedure, however, also severely reduced food intake and weight gains during pregnancy which might confound drug effects with nutritional deficiency. The drug can be injected in doses which produce plasma levels well within the therapeutic range for humans. Under these conditions the drug is less detrimental to the progression of pregnancy, however, the higher doses can increase neonatal mortality and **reduce body weight** of surviving offspring. Although mortality and body weight are not adversely effected by lower doses, changes are still apparent in the behavior as well as several biochemical parameters. Fostering studies on animals suggest that the effects of maternal injections of phenobarbital on offspring are due to the IN UTERO exposure rather than postnatal maternal factors and that effects produced by fostering itself may be confounded with the drug effects.
ACCESSION NUMBER: 87065820 MEDLINE
DOCUMENT NUMBER: 87065820 PubMed ID: 3785754
TITLE: Phenobarbital during pregnancy in mouse and man.
AUTHOR: Middaugh L D
SOURCE: NEUROTOXICOLOGY, (1986 Summer) 7 (2) 287-301.
Journal code: 7905589. ISSN: 0161-813X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198701
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19970203
Entered Medline: 19870121

L5 ANSWER 104 OF 621 MEDLINE
TI Diurnal patterns in homecage behavior of rats after acute exposure to triethyltin.
AB Diurnal patterns of eating, drinking, locomotor activity, and rearing in

male Fischer-344 rats were examined for 11 days after a single oral dose of triethyltin bromide (TET) at 0, 1.5, 3, or 5 mg/kg. The 5 mg/kg group exhibited a time-related drop in food consumption and body weight until 3 of 10 rats were sacrificed moribund 11 days after dosing. Doses of 1.5 and 3 mg/kg TET did not **reduce body weight** or consumption of food and water. In contrast, food consumption was significantly increased 7 and 11 days after 3 mg/kg TET, and diurnal patterns of eating and drinking were disrupted 7 days after 3 and 5 mg/kg TET. A phase shift in licking patterns was induced by the high dose. Unlike trimethyltin (TMT), TET did not affect efficiency of eating. Diurnal patterns of both horizontal and vertical activity were disrupted at all dose levels on Day 2 after dosing; by 16 days after dosing, recovery was evident in all rats including those surviving 5 mg/kg TET. These results show that a near-lethal dose of TET produced a reversible syndrome of hypoactivity, aphagia, and weight loss similar to that seen after acute TMT; in the absence of the above signs, diurnal patterns of behavior revealed effects of TET at doses as low as 1.5 mg/kg; the magnitude of the effect depended on the time of day at which the response was measured; and TET did not produce the same effects on ingestive behaviors (polydipsia and reduced feeding efficiency) that were previously observed after acute TMT.

ACCESSION NUMBER: 87019896 MEDLINE
DOCUMENT NUMBER: 87019896 PubMed ID: 3764920
TITLE: Diurnal patterns in homecage behavior of rats after acute exposure to triethyltin.
AUTHOR: Bushnell P J; Evans H L
CONTRACT NUMBER: ES-00260 (NIEHS)
ES-03461 (NIEHS)
ES-2-5017 (NIEHS)
SOURCE: TOXICOLOGY AND APPLIED PHARMACOLOGY, (1986 Sep 30) 85 (3) 346-54.
Journal code: 0416575. ISSN: 0041-008X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198611
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19970203
Entered Medline: 19861113

L5 ANSWER 105 OF 621 MEDLINE

TI Cell proliferation in developing brain after brief exposure to nitrous oxide or halothane.

AB Several inhalant anesthetics, including nitrous oxide and halothane, are known to be antimitotic in a variety of developing tissues, but none has been tested for antimitotic activity in developing brain. Concern about the safety of these agents has centered around behavioral effects reported in humans and animals after early exposure. Because interference with cell production during CNS development is a sufficient cause for later behavioral abnormalities, it is important to know whether cell production in the nervous system is altered by these agents. Mice were exposed to either nitrous oxide (75% N2O and 25% O2) or halothane (0.5% halothane in 75% N2 and 25% O2) or a mixture of 75% N2 and 25% O2. Prenatal treatment groups were exposed for 6 h on the 14th day of gestation, while postnatal treatment groups were exposed for 4 h on the second day after birth. Treated and control animals were then killed immediately after exposure, or 12, 24, or 48 h later, to be evaluated for CNS mitotic activity. Each of the four anesthetic-exposed groups showed some deviations from normal mitosis, but only the postnatal nitrous oxide group showed the pattern of reduced cell proliferation followed by a rebound that is characteristic of many antimitotic teratogens. Although prenatal nitrous oxides' effects on the fetal brain were not clearly interpretable, it did delay development of blood, as has been reported by other investigators. Both nitrous oxide

and halothane significantly reduced body weight of fetuses in utero, but did not **reduce body weight** of neonates. The pattern of the body-weight effects suggests that they occur by some mechanism other than reduced cell production.(ABSTRACT TRUNCATED AT 250 WORDS)

ACCESSION NUMBER: 86240217 MEDLINE
DOCUMENT NUMBER: 86240217 PubMed ID: 2940944
TITLE: Cell proliferation in developing brain after brief exposure to nitrous oxide or halothane.
AUTHOR: Rodier P M; Aschner M; Lewis L S; Koeter H B
CONTRACT NUMBER: RRO-5403 (NCRR)
SOURCE: ANESTHESIOLOGY, (1986 Jun) 64 (6) 680-7.
Journal code: 1300217. ISSN: 0003-3022.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198606
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19860630

L5 ANSWER 106 OF 621 MEDLINE
TI Gonadal influences on adiposity.
AB Gonadal steroids affect energy balance and adiposity in a variety of mammalian species. For example, estradiol acts via multiple, redundant mechanisms to **reduce body weight** and adiposity. The steroid can act directly in the brain to decrease food intake and stimulate voluntary exercise. Sex hormones may act concurrently in non-neural peripheral tissues to alter metabolic processes and energy balance. White adipose tissue estrogen receptors may mediate estradiol-induced decreases in lipoprotein lipase activity and lipid storage. Finally, estradiol increases energy expenditure independent of any effects on voluntary exercise. Brown adipose tissue is a potential site for estradiol-induced thermogenesis.

ACCESSION NUMBER: 86058240 MEDLINE
DOCUMENT NUMBER: 86058240 PubMed ID: 4066126
TITLE: Gonadal influences on adiposity.
AUTHOR: Wade G N; Gray J M; Bartness T J
CONTRACT NUMBER: AM 20785 (NIADDK)
MH 00321 (NIMH)
NS 10873 (NINDS)
SOURCE: INTERNATIONAL JOURNAL OF OBESITY, (1985) 9 Suppl 1 83-92.
Journal code: 7703240. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198601
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860121

L5 ANSWER 107 OF 621 MEDLINE
TI Alcohol and obesity: a new look at high blood pressure and stroke. An epidemiological study in preventive neurology.
AB An investigation of the staff of a car assembly plant (3,351 persons) revealed a similarity between the change in relative body weight and diastolic blood pressure with age. There is a good temporal correlation between the course of alcohol consumption during life and the change of the relative body weight. German women had significantly less blood pressure for the same relative body weight than German men, and foreign employees had lower blood pressure than Germans. In both cases the main cause is the difference in alcohol consumption. Besides obesity and

hereditary factors, alcohol is the main cause of "essential" hypertension today. Epidemiological and experimental data indicate that there are two ways from alcohol to high blood pressure, a more direct one and an indirect one via obesity. Alcohol causes obesity via a change in metabolism (hyperinsulinism) rather than by higher caloric intake. In both ways alcohol is an important cause of stroke. To **reduce body weight** and blood pressure, a reduction of alcohol consumption should be recommended in addition to reduced caloric intake and increased physical activity as means of preventive neurology.

ACCESSION NUMBER: 85285229 MEDLINE
DOCUMENT NUMBER: 85285229 PubMed ID: 3896816
TITLE: Alcohol and obesity: a new look at high blood pressure and stroke. An epidemiological study in preventive neurology.
AUTHOR: Kornhuber H H; Lisson G; Suschka-Sauermann L
SOURCE: EUROPEAN ARCHIVES OF PSYCHIATRY AND NEUROLOGICAL SCIENCES, (1985) 234 (6) 357-62.
Journal code: 8411522. ISSN: 0175-758X.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198509
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850927

L5 ANSWER 108 OF 621 MEDLINE
TI Adipose tissue characteristics of ex-obese long-distance runners.
AB Mean fat cell diameter and lipolytic activities of adipose tissue were determined in five ex-obese runners (EOR) who had experienced a mean weight loss of 39.5 kg during the course of a long-distance running program. At the time of investigation, their mean weekly running distance was 95 km in which no diet manipulation was needed to maintain their body weight. Body fat was estimated to be 14.3 percent of their body weight. Despite the high amount of exercise, subjects were not able to **reduce body weight** any further. Their values were compared with those obtained in the two following groups (1): six elite long-distance runners (ER) having a lower percent body fat (percent fat = 9.5); (2) five sedentary controls (SC) who were paired for adiposity with EOR (percent fat = 14.4). All subjects were submitted to a biopsy of subcutaneous fat in the suprailiac region, in which mean fat cell diameter was 57.5, 74.5, and 86 microns in EOR, ER and SC respectively. Basal and epinephrine-stimulated lipolysis were similar in EOR and SC, while ER exhibited higher values. These results indicate that exercise-training can produce important weight losses when subjects are capable of tolerating high levels of energy expenditure for extended periods of time. Moreover, the reduced fat cell diameter for a higher adiposity in EOR as compared to ER suggests that resistance to further fat loss may occur when the fat cell size is markedly reduced. No deficit in maximal epinephrine stimulated lipolysis of isolated fat cells was found in EOR in comparison to SC subjects, but they failed to adapt like lean ER subjects.

ACCESSION NUMBER: 85181868 MEDLINE
DOCUMENT NUMBER: 85181868 PubMed ID: 6533088
TITLE: Adipose tissue characteristics of ex-obese long-distance runners.
AUTHOR: Tremblay A; Despres J P; Bouchard C
SOURCE: INTERNATIONAL JOURNAL OF OBESITY, (1984) 8 (6) 641-8.
Journal code: 7703240. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198506
ENTRY DATE: Entered STN: 19900320

Last Updated on STN: 19900320
Entered Medline: 19850606

L5 ANSWER 109 OF 621 MEDLINE

TI Treatment with vitamin D or its analogues does not change body weight or blood glucose level in postmenopausal women.

AB It has recently been stated that vitamin D analogues can **reduce body weight** in animal models. We evaluated the effect of treatment with vitamin D metabolites on the nutritional status of 238 postmenopausal women, who were participating in three double-blind placebo-controlled trials, the aim of which was to prevent or treat postmenopausal bone loss. After two years of treatment with vitamin D3, 2000 IU daily (n = 25), or 1 alpha OHD3, 0.25 micrograms daily (n = 23), or 1 year of treatment with 1,25 (OH)2D3, 0.25-0.50 micrograms daily (n = 40), no change in body weight or blood glucose level could be detected when compared to corresponding placebo treated groups (n = 150). It is concluded that the effect of vitamin D analogues on body weight is a phenomenon which is specific for an animal model and does not occur in man.

ACCESSION NUMBER: 85104032 MEDLINE
DOCUMENT NUMBER: 85104032 PubMed ID: 6549176
TITLE: Treatment with vitamin D or its analogues does not change body weight or blood glucose level in postmenopausal women.
AUTHOR: Nilas L; Christiansen C
SOURCE: INTERNATIONAL JOURNAL OF OBESITY, (1984) 8 (5) 407-11. Journal code: 7703240. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198503
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850301

L5 ANSWER 110 OF 621 MEDLINE

TI Thermogenic drugs for the treatment of obesity: sympathetic stimulants in animal models.

AB Thirty-three drugs known to stimulate the sympathetic nervous system have been screened for thermogenic properties. The results presented are for seven of them. The drugs were tested in five animal models of obesity (genetic (mice and rats), hypothalamic (mice) and dietary (mice and rats] as well as in lean mice. Energy-balance studies were undertaken using the comparative-carcass technique as well as by measurement of daily oxygen consumption. All seven drugs in obese animals tended to **reduce body-weight** and fat without loss of body protein: they acted by increasing metabolic rate without increasing food intake. They were much less effective in lean animals. These findings lend support to the concept that obesity is due to a diminished activity of the sympathetic nervous system. Differences in the effectiveness of the drugs are discussed in relation to differences between the animal models of obesity. Ephedrine and tranylcypromine were found to be the most effective drugs in this series of experiments and a prima facie case is made for human clinical trials.

ACCESSION NUMBER: 85000396 MEDLINE
DOCUMENT NUMBER: 85000396 PubMed ID: 6477859
TITLE: Thermogenic drugs for the treatment of obesity: sympathetic stimulants in animal models.
AUTHOR: Dulloo A G; Miller D S
SOURCE: BRITISH JOURNAL OF NUTRITION, (1984 Sep) 52 (2) 179-96. Journal code: 0372547. ISSN: 0007-1145.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198411
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19841102

L5 ANSWER 111 OF 621 MEDLINE
TI BRL 20459, a novel topically active non-steroidal anti-inflammatory drug.
AB BRL 20459 is a novel compound which displays anti-inflammatory activity when applied topically in the croton oil and cantharadin rat ear inflammation models. The compound does not inhibit uv-induced erythema in the guinea-pig or granuloma formation in the cotton pellet test in the rat. BRL 20459 does not inhibit prostaglandin synthesis nor does it interact with corticosteroid receptors in the thymus. In contrast to hydrocortisone, BRL 20459 did not cause thymus involution or **reduce body weight** gain in rats. BRL 20459 would seem to have a different mechanism of action to hydrocortisone, but this mechanism is as yet unknown.
ACCESSION NUMBER: 84242291 MEDLINE
DOCUMENT NUMBER: 84242291 PubMed ID: 6145767
TITLE: BRL 20459, a novel topically active non-steroidal anti-inflammatory drug.
AUTHOR: Green A P; Mangan F R; Thomson M J; Randall K E; Boyle E A
SOURCE: JOURNAL OF PHARMACY AND PHARMACOLOGY, (1984 May) 36 (5) 314-7.
Journal code: 0376363. ISSN: 0022-3573.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198408
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19950206
Entered Medline: 19840817

L5 ANSWER 112 OF 621 MEDLINE
TI Drugs in muscular dystrophy of the chicken: corticosterone-21-acetate.
AB In a previous series of 22-day evaluations of 31 compounds, only corticosterone-21-acetate (C-21-A) increased righting ability of genetically dystrophic chickens to a greater extent than the standard of comparison, methysergide maleate. In the present study, C-21-A was subjected to longer-term trials of up to 48 days, and additional signs of the myopathy were examined. The highest doses of C-21-A increased righting ability for the duration of the trials, decreased the typically elevated plasma levels of creatine kinase (CK) activity by more than 80%, and improved morphology of the dystrophic pectoralis major muscle at the light microscopic level. The major adverse effect of C-21-A, reduction of body weight, was consistently observed at the relatively high doses needed to increase righting ability. That alone, however, could not account for increased righting ability, and plasma CK activity was decreased even at doses that did not **reduce body weight**. The results show that C-21-A is the most effective compound yet tested in this system and, perhaps more significantly, provides the first evidence that it is possible to identify compounds that improve muscle morphology in a hereditary myopathy using a short-term, step-wise system.
ACCESSION NUMBER: 84191312 MEDLINE
DOCUMENT NUMBER: 84191312 PubMed ID: 6717489
TITLE: Drugs in muscular dystrophy of the chicken: corticosterone-21-acetate.
AUTHOR: Entrikin R K; Patterson G T; Wilson B W
CONTRACT NUMBER: G008300078
SOURCE: MUSCLE AND NERVE, (1984 Feb) 7 (2) 130-6.
Journal code: 7803146. ISSN: 0148-639X.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198405
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840530

L5 ANSWER 113 OF 621 MEDLINE

TI Comparative effects of estradiol stereoisomers on pimozide-induced catalepsy, locomotor activity and body-weight in the rat.

AB The effects of 17-alpha and 17-beta estradiol were compared at three dose levels on locomotor activity, pimozide-induced catalepsy, and changes in body weight. At 10 micrograms/kg/day they increased locomotor activity to a similar degree but at 5 and 1 microgram/kg/day the beta form was mor effective. However the alpha isomer failed to potentiate catalepsy, or **reduce body weight**, even at the highest dose whereas 17-beta estradiol did both. From these and other results it is suggested that estradiol might act on intracellular receptors and not by changing catecholamine metabolism.

ACCESSION NUMBER: 84070967 MEDLINE
DOCUMENT NUMBER: 84070967 PubMed ID: 6685881
TITLE: Comparative effects of estradiol stereoisomers on pimozide-induced catalepsy, locomotor activity and body-weight in the rat.
AUTHOR: Johnson N J; Stevens R
SOURCE: PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, (1983 Nov) 19 (5) 801-5.
Journal code: 0367050. ISSN: 0091-3057.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198401
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19840127

L5 ANSWER 114 OF 621 MEDLINE

TI Thermogenic and antiobesity activity of a novel beta-adrenoceptor agonist (BRL 26830A) in mice and rats.

AB The effects of a novel compound, BRL 26830A, on energy balance in normal and obese mice have been investigated. BRL 26830A reduced body weight or weight gain in genetically (ob/ob), goldthioglucose, and cafeteria diet obese mice and genetically obese (fa/fa) Zucker rats. Weight reduction was due to reduced body lipid content. BRL 26830A caused little or no reduction in food intake in these animals but it increased metabolic rate and in genetically obese mice this thermic effect was increased by repeat dosing. BRL 26830A did not **reduce body weight** gain in the lean counterparts of the genetically obese animals. Its thermic effect was smaller in the lean than the genetically obese mice and it caused an increase in food intake in the lean mice. The thermic effect of BRL 26830A was inhibited by dl- but not d-propranolol. BRL 26830A largely overcame the depression in metabolic rate caused by fasting.

ACCESSION NUMBER: 84020934 MEDLINE
DOCUMENT NUMBER: 84020934 PubMed ID: 6137948
TITLE: Thermogenic and antiobesity activity of a novel beta-adrenoceptor agonist (BRL 26830A) in mice and rats.
AUTHOR: Arch J R; Ainsworth A T
SOURCE: AMERICAN JOURNAL OF CLINICAL NUTRITION, (1983 Oct) 38 (4) 549-58.
Journal code: 0376027. ISSN: 0002-9165.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198311
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831123

L5 ANSWER 115 OF 621 MEDLINE
TI Parallel adrenal and renal abnormalities in young patients with essential hypertension.
AB To determine whether the previously described abnormalities in adrenal secretion and renal blood flow in essential hypertension are associated, we examined the responses to the relevant systems in 18 patients with essential hypertension. Young patients, under 30 years of age, were studied to minimize the likelihood that the phenomena were secondary to long-standing hypertension. To achieve a wide span of sodium balance, studies were performed during a high (200 mEq) sodium intake, a restricted (10 mEg) sodium intake and a restricted sodium intake supplemented by a further short-term diuretic-induced volume deficit (furosemide, 180 to 300 mg, to **reduce body weight** by 1 to 1.5 kg).
The indexes measured included cardiac output (indocyanine green indicator dilution), plasma volume (125 I albumin space), renal blood flow (radioxenon transit), plasma renin activity and aldosterone levels and aldosterone secretory rate. All of these variables, with the exception of blood pressure and total peripheral resistance, were within the normal range during the two diets. However, the aldosterone secretory response to diuretic-induced volume depletion on a low-sodium diet was clearly blunted in nine subjects. These nine subjects (abnormal responders) had a virtually absent aldosterone increment (23 +/- 34 micrograms per 24 hours) compared with the normal responders (502 %/- 70 micrograms per 24 hours). In addition, renal blood flow was significantly higher in these same nine subjects during both a high sodium intake (434 +/- 19 versus 342 +/- 26 ml/100 g per minute) and a restricted sodium intake /446 +/- 11 versus 285 +/- 39 ml/100 g per minute). Yet, there were no significant differences between these two groups in sodium or potassium balance, blood pressure, plasma volume, cardiac index or plasma renin activity during a high or low sodium intake. Normally, control of both aldosterone release by the adrenal and renal perfusion is dominated by angiotensin; an apparently blunted response of both systems suggests that there may be a generalized abnormality in the way angiotensin interacts with its target tissues in many young patients with essential hypertension.
ACCESSION NUMBER: 82227700 MEDLINE
DOCUMENT NUMBER: 82227700 PubMed ID: 7091162
TITLE: Parallel adrenal and renal abnormalities in young patients with essential hypertension.
AUTHOR: Williams G H; Tuck M L; Sullivan J M; Dluhy R G; Hollenberg N K
CONTRACT NUMBER: GM 18674 (NIGMS)
HE 05832
HL 14944 (NHLBI)
SOURCE: AMERICAN JOURNAL OF MEDICINE, (1982 Jun) 72 (6) 907-14.
Journal code: 0267200. ISSN: 0002-9343.
Report No.: NASA-82227700.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Space Life Sciences
ENTRY MONTH: 198208
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19970203
Entered Medline: 19820807

L5 ANSWER 116 OF 621 MEDLINE

TI Morphometric effects of postnatal lead exposure on hippocampal development of the 15-day-old rat.

AB Neurotoxic sequelae of developmental lead exposure suggest that the hippocampus may be affected. Therefore, rats received low-level exposure via the milk of dams drinking 0.2% lead acetate beginning at parturition, and mid-dorsal sections of the hippocampus and dentate gyrus (DG) from 15-day-old pups were examined by light and electron microscopy. Lead exposure did not **reduce body weight** nor produce obviously abnormal vascularity or signs of cytotoxicity in the hippocampal formation, and total numbers per section of dentate granule cells or CA3 pyramidal cells were not reduced. On the other hand, lead exposure reduced neuropil development as evidenced both by reduced areas of the dentate hilus and dentate infrapyramidal stratum moleculare and by increased number of hilar CA3 pyramidal cells per unit area. Also, lead exposure reduced numbers of several types of synaptic profiles per unit area in the suprapyramidal mossy fiber zone. Complex invaginated (CI) profiles, assumed to be mature mossy fiber boutons, were characterized by multiple membrane densities and deep invaginations around dendritic spines of pyramidal cells. Complex noninvaginated (CN) boutons exhibited bag-like profiles with multiple membrane densities. Smaller, less numerous, simple (S) profiles contacted either dendritic trunks (ST) or spines (SS). Lead exposure reduced the numerical density of any of the profiles in the deep (close to stratum pyramidale) part of the proximal (close to DG) region of the suprapyramidal mossy fiber zone, but did not alter the numerical density of any of the profiles in the superficial (distal to stratum pyramidale) parts of either proximal or distal (close to CA1) regions. Average size of CN profiles in the distal region was increased by lead exposure. The pattern of effects suggests that low-level lead exposure during development preferentially affects later developing structures within the hippocampal formation, rather than affecting mature structures.

ACCESSION NUMBER: 82183636 MEDLINE
DOCUMENT NUMBER: 82183636 PubMed ID: 7074364
TITLE: Morphometric effects of postnatal lead exposure on hippocampal development of the 15-day-old rat.
AUTHOR: Campbell J B; Woolley D E; Vijayan V K; Overmann S R
CONTRACT NUMBER: ES-01503 (NIEHS)
SOURCE: BRAIN RESEARCH, (1982 Apr) 255 (4) 595-612.
Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198207
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19970203
Entered Medline: 19820722

L5 ANSWER 117 OF 621 MEDLINE

TI Truncal vagotomy in morbid obesity.

AB Vagotomy has been shown to **reduce body weight** in several species of experimental animals. Due to the relative safety and simplicity of the procedure and the long-clinical evaluation of vagotomy in ulcer disease, truncal vagotomy without drainage has been performed in a series of 21 morbidly obese patients. The mean maximum body weight was 12.8 +/- 3 kg (s.e.). In the 14 patients observed for 12-40 months, the mean weight decrease is 20 +/- 4 kg (range: 0-51). Apart from lesion of the oesophagus in one patient, there have been no operative complications. In one 45-year-old patient sudden death due to myocardial fibrosis occurred three years after the operation. Four patients have had short episodes of diarrhea, and vomiting has occurred in two patients who "tested the limits'. There is no evidence of gastric dilatation or ulcers, yet gastric stasis is prevalent. Three patients are failures, two not having reduced and the third regaining 28 of her initial

31 kg weight loss postoperatively. Five patients have participated in programs for weight reduction in which they claim greater ease in complying than before operation, due to the characteristic lack of hunger sensations in all of the successful patients. The mechanisms for weight reduction after vagotomy are not known, yet seem to involve other factors than delayed gastric emptying of solids. Longer follow-up is necessary for evaluation of this procedure in the treatment of morbid obesity.

ACCESSION NUMBER: 82075012 MEDLINE
DOCUMENT NUMBER: 82075012 PubMed ID: 7309328
TITLE: Truncal vagotomy in morbid obesity.
AUTHOR: Kral J G; Gortz L
SOURCE: INTERNATIONAL JOURNAL OF OBESITY, (1981) 5 (4) 431-5.
Journal code: 7703240. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198202
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19820222

L5 ANSWER 118 OF 621 MEDLINE
TI Effects of maturation and aging on behavioral responses to haloperidol in the rat.
AB Male Sprague-Dawley rats were evaluated between ages 18 and 825 days for responses to doses of haloperidol (0 and 0.05-10 mg/kg, IP). Catalepsy, ptosis, and inhibition of general motor activity showed steady decreases in sensitivity to the drug with age during the first 1.5 years of maturation, while rats older than 1.5 years had strikingly increased sensitivity to the activity-inhibiting and cataleptic effects of the drug. The efficacy of haloperidol on all tests in 110-day old rats was indistinguishable whether food was available continuously, or restricted to **reduce body weight** by 55%, indicating
that the effects of maturation are due to aging and not to increasing body weight. The effects may be due to altered drug metabolism or altered sensitivity of the central nervous system to neuroleptic agents. Clinical impressions too, indicate that immature and elderly patients are more sensitive to these and other psychotropic drugs than are young adults.

ACCESSION NUMBER: 81224434 MEDLINE
DOCUMENT NUMBER: 81224434 PubMed ID: 6787640
TITLE: Effects of maturation and aging on behavioral responses to haloperidol in the rat.
AUTHOR: Campbell A; Baldessarini R J
SOURCE: PSYCHOPHARMACOLOGY, (1981) 73 (3) 219-22.
Journal code: 7608025. ISSN: 0033-3158.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198108
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810810

L5 ANSWER 119 OF 621 MEDLINE
TI Fat cell number, resting metabolic rate, mean heart rate, and insulin elevation while seeing and smelling food as predictors of slimming.
AB The explanatory value of total fat cell number, resting metabolic rate, mean heart rate during sleep, and peripheral insulin while seeing and smelling food were examined in relation to weight reduction in 19 obese women on a 1100-kcal/day diet. The insulin response while seeing and smelling food was expressed as the insulin area (mU x min x 1(-1)) over the baseline level. Food was presented in front of the patient for 5 min.

Insulin was determined in short intervals 20 min before and 20 min after start of food presentation. Fat cell number, resting metabolic rate, and mean heart rate during sleep were determined with standard techniques. All patients went through a period of weight loss, a plateau phase, and a period of weight regain. Body weight, fat cell number, resting metabolic rate, and/or heart rate correlated significantly with degree and rate of weight loss, duration of plateau phase, and rate of regain. In multiple regression analysis fat cell number and resting metabolic rate explained 81% of the variance for weight loss, 66% for rate of regain, and 29% for duration. For duration, only fat cell number contributed significantly. The variance of rate of weight loss was explained up to 49% by metabolic rate and insulin response while seeing and smelling food. The possibility of predicting weight reduction to a certain target weight is of great practical importance since the patients can obtain a realistic goal for their efforts to **reduce body weight**.
Hopefully systematic investigations of factors associated with the inability of obese subjects to maintain weight reduction will improve treatment in the future.

ACCESSION NUMBER: 81051692 MEDLINE
DOCUMENT NUMBER: 81051692 PubMed ID: 7001173
TITLE: Fat cell number, resting metabolic rate, mean heart rate, and insulin elevation while seeing and smelling food as predictors of slimming.
AUTHOR: Krotkiewski M; Garellick G; Sjostrom L; Persson G; Bjuro T; Sullivan L
SOURCE: METABOLISM: CLINICAL AND EXPERIMENTAL, (1980 Oct) 29 (11) 1003-12.
Journal code: 0375267. ISSN: 0026-0495.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 198101
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810126

L5 ANSWER 120 OF 621 MEDLINE
TI [Studies on the diuretic effects of etozolin (Elkapin) in heart failure - a comparison with the loop diuretic agent furosemide (author's transl)]. Etozolin (Elkapin) in der Behandlung der Herzinsuffizienz. Ein Vergleich mit der Wirkung des Schleifendiuretikums furosemid.
AB In 115 randomized patients with left and/or right ventricular failure, the effect of the new diuretic Etozolin (800 mg p.o.) (n = 55) is compared with that of the loop diuretic Furosemide (80 mg p.o.) (n = 60). Results: 1. The increased diuresis after Etozolin remains constant during the entire trial period. Furosemid initially induces a more intense diuresis. 2. Analysis of the action profile shows that the higher daily urinary output following Furosemide is due to a more intense diuresis in the first fractions of the day. The action of Etozolin is more constant and lasts into the evening. 3. Both substances **reduce body weight** to the same extent. 4. Heart rate and arterial blood pressure decline significantly during trial. 5. Etozolin induces lesser electrolyte elimination than Furosemide in the initial phase of the trial. Potassium elimination values, in particular, remain below Na+ and Cl- elimination values for both substances. 6. During the trial period both substances had no significant effect on blood and liver values, serum electrolytes, creatinine, urea and uric acid. Etozolin may be classified as a diuretic similar to a thiazide derivative in its 24-h profile but behaves like a loop diuretic in its diuretic effect.

ACCESSION NUMBER: 79221068 MEDLINE
DOCUMENT NUMBER: 79221068 PubMed ID: 460026
TITLE: [Studies on the diuretic effects of etozolin (Elkapin) in heart failure - a comparison with the loop diuretic agent

furosemide (author's transl)].
Etozolin (Elkapin) in der Behandlung der Herzinsuffizienz. Ein Vergleich mit der Wirkung des Schleifendiuretikums furosemid.

AUTHOR: Biamino G; Kopp H; Rudroff W; Schneider B
SOURCE: MEDIZINISCHE KLINIK, (1979 Apr 20) 74 (16) 624-30.
Journal code: 0376637. ISSN: 0025-8458.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197909
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19980206
Entered Medline: 19790925

L5 ANSWER 121 OF 621 MEDLINE
TI Some effects of reduced energy intake on the development of anaplasmosis in Bos indicus cross steers.
AB Some effects of the plane of nutrition on the development of anaplasmosis in Brahman cross steers were investigated. Batches of 39 and 30 Brahman cross steers, aged approximately 27 months were each divided by stratified randomisation into 4 groups of similar mean PCV and body weight. Two similar experiments, designated A and B were conducted. Groups 1 and 2 were fed a ration of lucerne chaff at the rate of 1 M Cal ME/80 kg live weight/day for 8 weeks aimed to **reduce body weight** by approximately 5 kg/week. Animals in groups 3 and 4 were fed a ration for the same period aimed to increase body weight by approximately 2 kg/week. Groups 1 and 3 were then inoculated with approximately 10(10) Anaplasma marginale infected erythrocytes and the effects of the subsequent infections during the clinical and recovery phases were examined by measuring humoral antibody response, packed cell volume, parasitaemia and body weight. Groups 2 and 4 were uninfected controls. Anaplasmosis, as measured by three responses, was less severe in the starved animals of group 1. Significant differences in packed cell volume and parasitaemia were detected for short periods between the infected groups 1 and 3. Anaplasmosis caused losses of 6.2% and 5.9% in the mean body weight of group 3 animals in experiments A and B respectively. Most of this loss occurred during the clinical phase of the disease. The disease caused no apparent loss of weight in the infected animals of group 1.

ACCESSION NUMBER: 78256555 MEDLINE
DOCUMENT NUMBER: 78256555 PubMed ID: 687262
TITLE: Some effects of reduced energy intake on the development of anaplasmosis in Bos indicus cross steers.
AUTHOR: Wilson A J; Trueman K F
SOURCE: AUSTRALIAN VETERINARY JOURNAL, (1978 Mar) 54 (3) 121-4.
Journal code: 0370616. ISSN: 0005-0423.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197810
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781018

L5 ANSWER 122 OF 621 MEDLINE
TI Gaining and losing weight in athletics.
AB Participants in many sports, such as wrestling, gymnastics, and light-weight crew, attempt to **reduce body weight** to achieve a maximum ratio of muscle strength to body

weight. Such weight reduction should result only from reduction in excess body fat. In most instances, weight reduction should be achieved at a rate of no more than 1 kg a week, through a modest reduction in diet and a moderate increase in exercise. More rapid weight reduction by starvation and dehydration compromises strength and endurance. Athletes attempting to gain weight should increase weight as muscle mass, not fat. Muscle mass is increased only through muscle work supported by an appropriate increase in food intake. No food, vitamin, drug, or hormone will increase muscle mass. It is recommended that the high caloric diet required to support muscle growth from increased work should be low in animal fats and cholesterol.

ACCESSION NUMBER:	76217822 MEDLINE
DOCUMENT NUMBER:	76217822 PubMed ID: 947010
TITLE:	Gaining and losing weight in athletics.
AUTHOR:	Smith N J
SOURCE:	JAMA, (1976 Jul 12) 236 (2) 149-51. Journal code: 7501160. ISSN: 0098-7484.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:	197609
ENTRY DATE:	Entered STN: 19900313 Last Updated on STN: 19900313 Entered Medline: 19760901

L5 ANSWER 123 OF 621 USPATFULL

TI Secreted and transmembrane polypeptides and nucleic acids encoding the same

AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

ACCESSION NUMBER:	2003:112862 USPATFULL
TITLE:	Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S):	Ashkenazi, Avi J., San Mateo, CA, UNITED STATES Baker, Kevin P., Darnestown, MD, UNITED STATES Botstein, David, Belmont, CA, UNITED STATES Desnoyers, Luc, San Francisco, CA, UNITED STATES Eaton, Dan L., San Rafael, CA, UNITED STATES Ferrara, Napoleone, San Francisco, CA, UNITED STATES Fong, Sherman, Alameda, CA, UNITED STATES Gerber, Hanspeter, San Francisco, CA, UNITED STATES Gerritsen, Mary E., San Mateo, CA, UNITED STATES Goddard, Audrey, San Francisco, CA, UNITED STATES Godowski, Paul J., Hillsborough, CA, UNITED STATES Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES Gurney, Austin L., Belmont, CA, UNITED STATES Kljavin, Ivar J., Lafayette, CA, UNITED STATES Napier, Mary A., Hillsborough, CA, UNITED STATES Pan, James, Belmont, CA, UNITED STATES Paoni, Nicholas F., Belmont, CA, UNITED STATES Roy, Margaret Ann, San Francisco, CA, UNITED STATES Stewart, Timothy A., San Francisco, CA, UNITED STATES Tumas, Daniel, Orinda, CA, UNITED STATES Watanabe, Colin K., Moraga, CA, UNITED STATES Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES Wood, William I., Hillsborough, CA, UNITED STATES

	Zhang, Zemin, Foster City, CA, UNITED STATES		
PATENT ASSIGNEE(S):	Genentech, Inc. (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003077594	A1	20030424
APPLICATION INFO.:	US 2001-993583	A1	20011114 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING		
	NUMBER	DATE	
PRIORITY INFORMATION:	WO 1997-US20069	19971105	
	WO 1998-US19330	19980916	
	WO 1998-US19437	19980917	
	WO 1998-US21141	19981007	
	WO 1998-US25108	19981201	
	WO 1999-US106	19990105	
	WO 1999-US5028	19990308	
	WO 1999-US12252	19990602	
	WO 1999-US21090	19990915	
	WO 1999-US21547	19990915	
	WO 1999-US28313	19991130	
	WO 1999-US28301	19991201	
	WO 1999-US28634	19991201	
	WO 1999-US30095	19991216	
	WO 1999-US30911	19990220	
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	WO 2000-US7377	20000320	
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	WO 2001-US21735	20010709	
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US	1998-113296P	19981222 (60)
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US	1999-141037P	19990623 (60)
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	US 1999-145698P 19990726 (60) US 1999-146222P 19990728 (60) US 1999-149396P 19990817 (60) US 1999-158663P 19991008 (60) US 2000-213637P 20000623 (60) US 2000-230978P 20000907 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS:	118
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	330 Drawing Page(s)
LINE COUNT:	32252

L5 ANSWER 124 OF 621 USPATFULL

TI Secreted and transmembrane polypeptides and nucleic acids encoding the same

AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

ACCESSION NUMBER:	2003:112861 USPATFULL
TITLE:	Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S):	Ashkenazi, Avi J., San Mateo, PA, UNITED STATES Baker, Kevin P., Darnestown, MD, UNITED STATES Botstein, David, Belmont, CA, UNITED STATES Desnoyers, Luc, San Francisco, CA, UNITED STATES Eaton, Dan L., San Rafael, CA, UNITED STATES Ferrara, Napoleone, San Francisco, CA, UNITED STATES Fong, Sherman, Alameda, CA, UNITED STATES Gerber, Hanspeter, San Francisco, CA, UNITED STATES Gerritsen, Mary E., San Mateo, CA, UNITED STATES Goddard, Audrey, San Francisco, CA, UNITED STATES Godowski, Paul J., Hillsborough, CA, UNITED STATES Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES Gurney, Austin L., Belmont, CA, UNITED STATES Kljavin, Ivar J., Lafayette, CA, UNITED STATES Napier, Mary A., Hillsborough, CA, UNITED STATES Pan, James, Belmont, CA, UNITED STATES Paoni, Nicholas F., Belmont, CA, UNITED STATES Roy, Margaret Ann, San Francisco, CA, UNITED STATES Stewart, Timothy A., San Francisco, CA, UNITED STATES Tumas, Daniel, Orinda, CA, UNITED STATES Watanabe, Colin K., Moraga, CA, UNITED STATES Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES Wood, William I., Hillsborough, CA, UNITED STATES Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S):	Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003077593	A1	20030424
APPLICATION INFO.:	US 2001-989328	A1	20011119 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

NUMBER	DATE

PRIORITY INFORMATION:

WO 1997-US20069	19971105
WO 1998-US19330	19980916
WO 1998-US19437	19980917
WO 1998-US21141	19981007
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US 1999-158663P	19991008 (60)
US 2000-213637P	20000623 (60)
US 2000-230978P	20000907 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610

NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32495

L5 ANSWER 125 OF 621 USPATFULL
TI METHODS OF IDENTIFYING OF SCREENING FOR AGENTS THAT BINDS THE OB-Re
AB This invention provides an isolated nucleic acid encoding a polypeptide, a purified polypeptide, vectors comprising isolated nucleic acid encoding a polypeptide, cells comprising such vectors, antibodies directed to a polypeptide, nucleic acid probes useful for detecting nucleic acid encoding a polypeptide, antisense oligonucleotides complementary to unique sequences of nucleic acid encoding a polypeptide, nonhuman transgenic animals which express DNA encoding a normal or a mutant polypeptide, methods of isolating a polypeptide, methods of treatment eating disorders as well as methods of determining binding of compounds to polypeptides.

ACCESSION NUMBER: 2003:106916 USPATFULL
TITLE: METHODS OF IDENTIFYING OF SCREENING FOR AGENTS THAT BINDS THE OB-Re
INVENTOR(S): ADHAM, NIKA, RIDGEWOOD, NJ, UNITED STATES
BOROWSKY, BETH, MONTCLAIR, NJ, UNITED STATES
LEVENS, NIGEL, ALLSCHWIL, SWITZERLAND
SKODA, RADEK C., BASEL, SWITZERLAND

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073829	A1	20030417
APPLICATION INFO.:	US 1998-116676	A1	19980716 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JOHN P WHITE, COOPER & DUNHAM, 1185 AVENUE OF THE AMERICAS, NEW YORK, NY, 10036
NUMBER OF CLAIMS: 207
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3245

L5 ANSWER 126 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:106896 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
Eaton, Dan L., San Rafael, CA, UNITED STATES
Ferrara, Napoleone, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Gerber, Hanspeter, San Francisco, CA, UNITED STATES
Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES

Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES

PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073809	A1	20030417
APPLICATION INFO.:	US 2001-990427	A1	20011114 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1997-US20069	19971105
	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
	WO 1998-US21141	19981007
	WO 1998-US25108	19981201
	WO 1999-US106	19990105
	WO 1999-US5028	19990308
	WO 1999-US12252	19990602
	WO 1999-US21090	19990915
	WO 1999-US21547	19990915
	WO 1999-US28313	19991130
	WO 1999-US28301	19991201
	WO 1999-US28634	19991201
	WO 1999-US30095	19991216
	WO 1999-US30911	19990220
	WO 2000-US219	20000105
	WO 2000-US376	20000106
	WO 2000-US3565	20000211
	WO 2000-US4341	20000218
	WO 2000-US4414	20000222
	WO 2000-US4914	20000224
	WO 2000-US5004	20000224
	WO 2000-US5841	20000302
	WO 2000-US6319	20000310
	WO 2000-US6884	20000315
	WO 2000-US7377	20000320
	WO 2000-US8439	20000330
	WO 2000-US13358	20000515
	WO 2000-US14042	20000522
	WO 2000-US15264	20000602
	WO 2000-US13705	20000517
	WO 2000-US14941	20000530
	WO 2000-US20710	20000728
	WO 2000-US22031	20000811
	WO 2000-US23522	20000823
	WO 2000-US23328	20000824
	WO 2000-US30952	20001108
	WO 2000-US32678	20001201

WO 2001-US6520	20010228	
WO 2001-US17800	20010601	
WO 2001-US19692	20010620	
WO 2001-US21066	20010629	
WO 2001-US21735	20010709	
US 1997-49787P	19970616	(60)
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US 1999-146222P	19990728	(60)
US 1999-149396P	19990817	(60)
US 1999-158663P	19991008	(60)
US 2000-213637P	20000623	(60)
US 2000-230978P	20000907	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32032
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 127 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:106179 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
Eaton, Dan L., San Rafael, CA, UNITED STATES
Ferrara, Napoleone, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Gerber, Hanspeter, San Francisco, CA, UNITED STATES
Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES

Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073090	A1	20030417
APPLICATION INFO.:	US 2001-990439	A1	20011116 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1997-US20069	19971105
	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
	WO 1998-US21141	19981007
	WO 1998-US25108	19981201
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	WO 1999-US28634	19991201
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	US 1999-143048P	19990707 (60)
	US 1999-144758P	19990720 (60)
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	US 2000-213637P	20000623 (60)
	US 2000-230978P	20000907 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 31979
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 128 OF 621 USPATFULL
TI Methods for treating disorders using plant extracts
AB The present invention provides materials and methods relating to mildly polar fluid extracts of plant materials, such as Artemisia plant species, useful in methods for treating diabetes and methods for modulating the activity of glucagon-like peptide-1 (GLP-1), and in methods for modulating phosphoenol pyruvate carboxykinase (PEPCK) activity in a diabetes-specific manner. The extracts are generally non-toxic and non-mutagenic and may be administered to diabetics with beneficial effect on blood glucose levels. The extracts may also be administered to non-diabetics without deleterious effect. The plants are easily grown with a minimum of time, labor, and cost. Extracts are inexpensively and quickly prepared without the need for fractionation to remove potentially deleterious compounds, and the extracts may be administered to mammals such as humans through various routes, in a variety of forms, and at convenient concentrations.

ACCESSION NUMBER: 2003:105911 USPATFULL
TITLE: Methods for treating disorders using plant extracts
INVENTOR(S): Ribnicky, David M., Plainsboro, NJ, UNITED STATES
Raskin, Ilya, Manalapan, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003072822	A1	20030417	
APPLICATION INFO.:	US 2002-232756	A1	20020830	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-316760P	20010831 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MARSHALL, GERSTEIN & BORUN, 6300 SEARS TOWER, 233 SOUTH WACKER, CHICAGO, IL, 60606-6357
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Page(s)
LINE COUNT: 1722

L5 ANSWER 129 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present

invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:100292 USPATFULL

TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same

INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
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Ferrara, Napoleone, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Gerber, Hanspeter, San Francisco, CA, UNITED STATES
Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES

PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069403	A1	20030410
APPLICATION INFO.:	US 2001-993748	A1	20011114 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1997-US20069	19971105
	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
	WO 1998-US21141	19981007
	WO 1998-US25108	19981201
	WO 1999-US106	19990105
	WO 1999-US5028	19990308
	WO 1999-US12252	19990602
	WO 1999-US21090	19990915
	WO 1999-US21547	19990915
	WO 1999-US28313	19991130
	WO 1999-US28301	19991201
	WO 1999-US28634	19991201
	WO 1999-US30095	19991216
	WO 1999-US30911	19990220
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	WO 2000-US376	20000106
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WO 2000-US7377	20000320	
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WO 2001-US17800	20010601	
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US 1999-149396P	19990817	(60)
US 1999-158663P	19991008	(60)
US 2000-213637P	20000623	(60)
US 2000-230978P	20000907	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32308
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 130 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:99542 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same

INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
Eaton, Dan L., San Rafael, CA, UNITED STATES
Ferrara, Napoleone, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Gerber, Hanspeter, San Francisco, CA, UNITED STATES
Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003068647	A1	20030410
APPLICATION INFO.:	US 2001-997542	A1	20011115 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1997-US20069	19971105
	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
	WO 1998-US21141	19981007
	WO 1998-US25108	19981201
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	WO 1999-US21090	19990915
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	WO 1999-US28313	19991130
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	WO 1999-US28634	19991201
	WO 1999-US30095	19991216
	WO 1999-US30911	19991220
	WO 2000-US219	20000105
	WO 2000-US376	20000106
	WO 2000-US3565	20000211
	WO 2000-US4341	20000218
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	WO 2000-US5841	20000302
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	WO 2000-US7377	20000320
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WO 2000-US20710	20000728	
WO 2000-US22031	20000811	
WO 2000-US23522	20000823	
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WO 2001-US19692	20010620	
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US 1998-113296P	19981222	(60)
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US 1999-149396P	19990817	(60)
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US 2000-213637P	20000623	(60)
US 2000-230978P	20000907	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32327
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 131 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:99518 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
Eaton, Dan L., San Rafael, CA, UNITED STATES
Ferrara, Napoleone, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Gerber, Hanspeter, San Francisco, CA, UNITED STATES

Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES

PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

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PATENT INFORMATION:	US 2003068623	A1	20030410
APPLICATION INFO.:	US 2001-993469	A1	20011114 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

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	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
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	WO 1999-US30095	19991216
	WO 1999-US30911	19991220
	WO 2000-US219	20000105
	WO 2000-US376	20000106
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	WO 2000-US13358	20000515
	WO 2000-US14042	20000522
	WO 2000-US15264	20000602
	WO 2000-US13705	20000517
	WO 2000-US14941	20000530
	WO 2000-US20710	20000728
	WO 2000-US22031	20000811
	WO 2000-US23522	20000823
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WO 2000-US32678	20001201	
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WO 2001-US21066	20010629	
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	US 1999-158663P	19991008 (60)
	US 2000-213637P	20000623 (60)
	US 2000-230978P	20000907 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610	
NUMBER OF CLAIMS:	118	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	330 Drawing Page(s)	
LINE COUNT:	32291	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 132 OF 621 USPATFULL

TI Secreted and transmembrane polypeptides and nucleic acids encoding the same

AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:93016 USPATFULL
TITLE:	Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S):	Ashkenazi, Avi J., San Mateo, CA, UNITED STATES Baker, Kevin P., Darnestown, MD, UNITED STATES Botstein, David, Belmont, CA, UNITED STATES Desnoyers, Luc, San Francisco, CA, UNITED STATES Eaton, Dan L., San Rafael, CA, UNITED STATES Ferrara, Napoleone, San Francisco, CA, UNITED STATES Fong, Sherman, Alameda, CA, UNITED STATES Gerber, Hanspeter, San Francisco, CA, UNITED STATES Gerritsen, Mary E., San Mateo, CA, UNITED STATES Goddard, Audrey, San Francisco, CA, UNITED STATES Godowski, Paul J., Hillsborough, CA, UNITED STATES Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES Gurney, Austin L., Belmont, CA, UNITED STATES Kljavin, Ivar J., Lafayette, CA, UNITED STATES Napier, Mary A., Hillsborough, CA, UNITED STATES

Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003064375	A1	20030403
APPLICATION INFO.:	US 2001-997857	A1	20011115 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1997-US20069	19971105
	WO 1998-US19330	19980916
	WO 1998-US19437	19980917
	WO 1998-US21141	19981007
	WO 1998-US25108	19981201
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	WO 1999-US21090	19990915
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	WO 1999-US28313	19991130
	WO 1999-US28301	19991201
	WO 1999-US28634	19991201
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	WO 1999-US30911	19990220
	WO 2000-US219	20000105
	WO 2000-US376	20000106
	WO 2000-US3565	20000211
	WO 2000-US4341	20000218
	WO 2000-US4414	20000222
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	WO 2000-US5004	20000224
	WO 2000-US5841	20000302
	WO 2000-US6319	20000310
	WO 2000-US6884	20000315
	WO 2000-US7377	20000320
	WO 2000-US8439	20000330
	WO 2000-US13358	20000515
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	WO 2000-US15264	20000602
	WO 2000-US13705	20000517
	WO 2000-US14941	20000530
	WO 2000-US20710	20000728
	WO 2000-US22031	20000811
	WO 2000-US23522	20000823
	WO 2000-US23328	20000824
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	WO 2000-US32678	20001201
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	WO 2001-US19692	20010620
	WO 2001-US21066	20010629
	WO 2001-US21735	20010709
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	US 2000-213637P	20000623 (60)
	US 2000-230978P	20000907 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610	
NUMBER OF CLAIMS:	118	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	330 Drawing Page(s)	
LINE COUNT:	32270	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 133 OF 621 USPATFULL

TI Secreted and transmembrane polypeptides and nucleic acids encoding the same

AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:86799 USPATFULL
TITLE:	Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S):	Ashkenazi, Avi J., San Mateo, CA, UNITED STATES Baker, Kevin P., Darnestown, MD, UNITED STATES Botstein, David, Belmont, CA, UNITED STATES Desnoyers, Luc, San Francisco, CA, UNITED STATES Eaton, Dan L., San Rafael, CA, UNITED STATES Ferrara, Napoleone, San Francisco, CA, UNITED STATES Fong, Sherman, Alameda, CA, UNITED STATES Gerber, Hanspeter, San Francisco, CA, UNITED STATES Gerritsen, Mary E., San Mateo, CA, UNITED STATES Goddard, Audrey, San Francisco, CA, UNITED STATES Godowski, Paul J., Hillsborough, CA, UNITED STATES Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES Gurney, Austin L., Belmont, CA, UNITED STATES Kljavin, Ivar J., Lafayette, CA, UNITED STATES Napier, Mary A., Hillsborough, CA, UNITED STATES Pan, James, Belmont, CA, UNITED STATES Paoni, Nicholas F., Belmont, CA, UNITED STATES Roy, Margaret Ann, San Francisco, CA, UNITED STATES Stewart, Timothy A., San Francisco, CA, UNITED STATES Tumas, Daniel, Orinda, CA, UNITED STATES Watanabe, Colin K., Moraga, CA, UNITED STATES Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES Wood, William I., Hillsborough, CA, UNITED STATES

Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003060407	A1	20030327
APPLICATION INFO.:	US 2001-990440	A1	20011114 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

PRIORITY INFORMATION:	NUMBER	DATE
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US 2000-230978P 20000907 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32295
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 134 OF 621 USPATFULL

TI Secreted and transmembrane polypeptides and nucleic acids encoding the same

AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:86228 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
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Gerritsen, Mary E., San Mateo, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Kljavin, Ivar J., Lafayette, CA, UNITED STATES
Napier, Mary A., Hillsborough, CA, UNITED STATES
Pan, James, Belmont, CA, UNITED STATES
Paoni, Nicholas F., Belmont, CA, UNITED STATES
Roy, Margaret Ann, San Francisco, CA, UNITED STATES
Stewart, Timothy A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059833	A1	20030327
APPLICATION INFO.:	US 2001-997440	A1	20011115 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28

Aug 2001, PENDING

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	US 2000-213637P	20000623 (60)
	US 2000-230978P	20000907 (60)
DOCUMENT TYPE:	Utility	

FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 330 Drawing Page(s)
LINE COUNT: 32459
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 135 OF 621 USPATFULL
TI Secreted and transmembrane polypeptides and nucleic acids encoding the same
AB The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:86227 USPATFULL
TITLE: Secreted and transmembrane polypeptides and nucleic acids encoding the same
INVENTOR(S): Ashkenazi, Avi J., San Mateo, CA, UNITED STATES
Baker, Kevin P., Darnestown, CA, UNITED STATES
Botstein, David, Belmont, CA, UNITED STATES
Desnoyers, Luc, San Francisco, CA, UNITED STATES
Eaton, Dan L., San Rafael, CA, UNITED STATES
Ferrara, Napoleone, San Francisco, CA, UNITED STATES
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Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Zhang, Zemin, Foster City, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059832	A1	20030327
APPLICATION INFO.:	US 2001-997349	A1	20011115 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-941992, filed on 28 Aug 2001, PENDING

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US 1998-83322P	19980428	(60)
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US 1998-87106P	19980528	(60)
US 1998-87607P	19980602	(60)
US 1998-87609P	19980602	(60)
US 1998-87759P	19980602	(60)
US 1998-87827P	19980603	(60)
US 1998-88021P	19980604	(60)
US 1998-88025P	19980604	(60)
US 1998-88026P	19980604	(60)
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US 1998-88217P 19980605 (60)
US 1998-88655P 19980609 (60)
US 1998-88734P 19980610 (60)
US 1998-88738P 19980610 (60)
US 1998-88742P 19980610 (60)
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US 1998-89801P 19980618 (60)
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US 1998-89908P 19980618 (60)
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US 1998-91633P 19980702 (60)
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US 1998-91978P 19980707 (60)
US 1998-91982P 19980707 (60)
US 1998-92182P 19980709 (60)
US 1998-92472P 19980710 (60)
US 1998-93339P 19980720 (60)

US 1998-94651P	19980730	(60)
US 1998-95282P	19980804	(60)
US 1998-95285P	19980804	(60)
US 1998-95302P	19980804	(60)
US 1998-95318P	19980804	(60)
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US 1998-95301P	19980804	(60)
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US 1998-96949P	19980818	(60)
US 1998-96950P	19980818	(60)
US 1998-96959P	19980818	(60)
US 1998-96960P	19980818	(60)
US 1998-97022P	19980818	(60)
US 1998-97141P	19980819	(60)
US 1998-97218P	19980820	(60)
US 1998-97661P	19980824	(60)
US 1998-97952P	19980826	(60)
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US 1998-97955P	19980826	(60)
US 1998-98014P	19980826	(60)
US 1998-97971P	19980826	(60)
US 1998-97974P	19980826	(60)
US 1998-97978P	19980826	(60)
US 1998-97986P	19980826	(60)
US 1998-97979P	19980826	(60)
US 1998-98525P	19980831	(60)
US 1998-100634P	19980916	(60)
US 1998-100858P	19980917	(60)
US 1998-113296P	19981222	(60)
US 1999-123957P	19990312	(60)
US 1999-141037P	19990623	(60)
US 1999-143048P	19990707	(60)
US 1999-144758P	19990720	(60)

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(FILE 'HOME' ENTERED AT 18:24:15 ON 25 APR 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, JICST-EPLUS, JAPIO, BIOSIS, BIOBUSINESS' ENTERED AT 18:24:55 ON 25 APR 2003

L1	80414	S	SOMATOSTATIN
L2	4567	S	L1 AND AGONIST
L3	1034	S	BODY WEIGHT REDUCTION
L4	181	S	L3 AND METHOD
L5	621	S	REDUCE BODY WEIGHT
L6	5	S	L5 AND L4

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L6 ANSWER 1 OF 5 MEDLINE
TI Randomized, double-blind trial of chitosan for **body weight reduction**.
AB BACKGROUND: Overweight and obesity is a prevalent and costly threat to public health. Compelling evidence links overweight and obesity with serious disorders such as cardiovascular diseases and diabetes. Dietary regimen are notoriously burdened with poor compliance. Chitosan is promoted in the US and other countries as an oral remedy to reduce fat absorption and has now been incorporated as a major constituent into several over-the-counter remedies. The primary aim of this study is to investigate the clinical effectiveness of oral chitosan for **body weight reduction**. METHODS: Thirty-four overweight volunteers were included in a randomized placebo-controlled double-blind trial. Subjects were assigned to receive either four capsules of chitosan or indistinguishable placebo twice daily for 28 consecutive days. Measurements were taken at baseline, after 14 and 28d of treatment. Subjects maintained their normal diet and documented the type and amount of food consumed. Adverse effects were assessed and compliance monitored. RESULTS: Data from 30 subjects were entered into an intention-to-treat analysis. After four weeks of treatment, body mass index, serum cholesterol, triglycerides, vitamin A, D, E and beta-carotene were not significantly different in subjects receiving chitosan compared to those receiving placebo. Vitamin K was significantly increased after four weeks in the chitosan group compared with placebo ($P<0.05$). Compliance was 91.5% and 96.0% for chitosan and placebo groups respectively. CONCLUSION: The above data suggest that chitosan in the administered dosage, without dietary alterations, does not **reduce body weight** in overweight subjects. No serious adverse effects were reported.

ACCESSION NUMBER: 1999296184 MEDLINE
DOCUMENT NUMBER: 99296184 PubMed ID: 10369493
TITLE: Randomized, double-blind trial of chitosan for **body weight reduction**.
AUTHOR: Pittler M H; Abbot N C; Harkness E F; Ernst E
CORPORATE SOURCE: Department of Complementary Medicine, School of Postgraduate Medicine and Health Sciences, University of Exeter, United Kingdom.
SOURCE: EUROPEAN JOURNAL OF CLINICAL NUTRITION, (1999 May) 53 (5) 379-81.
Journal code: 8804070. ISSN: 0954-3007.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990730
Last Updated on STN: 19990730
Entered Medline: 19990722

L6 ANSWER 2 OF 5 USPATFULL
TI OBG3 globular head and uses thereof for decreasing body mass
AB The present invention relates to the field of obesity research. Obesity is a public health problem that is serious and widespread. A compound, globular OBG3, has been identified that reduces weight gain in animals. This compound should be effective for reducing body mass and for treating obesity-related diseases and disorders. These obesity-related diseases and disorders include hyperlipidemias, atherosclerosis, diabetes, and hypertension.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:172323 USPATFULL
TITLE: OBG3 globular head and uses thereof for decreasing body mass
INVENTOR(S): Fruebis, Joachim, Cardiff, CA, UNITED STATES
Erickson, Mary Ruth, San Diego, CA, UNITED STATES
Yen-Potin, Frances, San Diego, CA, UNITED STATES
Bihain, Bernard, Encinitas, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002091080	A1	20020711
APPLICATION INFO.:	US 2001-909547	A1	20010719 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-776976, filed on 5 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-758055, filed on 10 Jan 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-176228P	20000114 (60)
	US 2000-198087P	20000413 (60)
	US 2000-229881P	20000901 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: John Lucas, Ph.D., J.D., GENSET CORP., 10665 Sorrento Valley Road, San Diego, CA, 92121-1609
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 24 Drawing Page(s)
LINE COUNT: 6195
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 5 USPATFULL
TI OBG3 globular head and uses thereof for decreasing body mass
AB The present invention relates to the field of obesity research. Obesity is a public health problem that is serious and widespread. A compound, globular OBG3, has been identified that reduces weight gain in animals. This compound should be effective for reducing body mass and for treating obesity-related diseases and disorders. These obesity-related diseases and disorders include hyperlipidemias, atherosclerosis, diabetes, and hypertension.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:112876 USPATFULL
TITLE: OBG3 globular head and uses thereof for decreasing body mass
INVENTOR(S): Fruebis, Joachim, Cardiff, CA, UNITED STATES
Erickson, Mary Ruth, San Diego, CA, UNITED STATES
Yen, Frances, San Diego, CA, UNITED STATES
Bihain, Bernard, Encinitas, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002058617	A1	20020516
APPLICATION INFO.:	US 2001-758055	A1	20010110 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-176228P	20000114 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
NUMBER OF CLAIMS: 9

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 28 Drawing Page(s)
LINE COUNT: 5839
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 5 USPATFULL
TI Regulators of PPARdelta (beta) and their use in the treatment of obesity and insulin resistance
AB Obesity is a common clinical problem in most developed nations and is also rapidly becoming a major health concern in developing nations. Overweight individuals frequently suffer from several metabolic disorders such as insulin resistance, type 2 diabetes and dyslipidemia.

This invention discloses proof of principle for the role PPAR.delta. (also known as .beta.) plays in the development of diet-induced obesity. In accordance with the present invention, a new **method** for treating obesity, insulin resistance and hyperlipidemia through administration of a pharmaceutical composition containing a chemical agent that antagonizes the function of PPAR.delta.(.beta.) protein, decreases PPAR.delta.(.beta.) gene expression and or transactivation of PPAR.delta.(.beta.) target gene expression is disclosed. This invention also proposes that obese, insulin resistant hyperlipidemic patients can be effectively treated with a combination of a PPAR.delta.(.beta.) antagonist with either an anti-diabetic agent or a lipid-lowering agent (or both).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:78702 USPATFULL
TITLE: Regulators of PPARdelta (beta) and their use in the treatment of obesity and insulin resistance
INVENTOR(S): Hariharan, Narayanan, Richboro, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002042359	A1	20020411
APPLICATION INFO.:	US 2001-909098	A1	20010719 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-219956P	20000720 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MARLA J MATHIAS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 824
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 5 USPATFULL
TI OBG3 globular head and uses thereof for decreasing body mass
AB The present invention relates to the field of obesity research. Obesity is a public health problem that is serious and widespread. A compound, globular OBG3, has been identified that reduces weight gain in animals. This compound should be effective for reducing body mass and for treating obesity-related diseases and disorders. These obesity-related diseases and disorders include hyperlipidemias, atherosclerosis, diabetes, and hypertension.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:67196 USPATFULL
TITLE: OBG3 globular head and uses thereof for decreasing body mass

INVENTOR(S): Fruebis, Joachim, Cardiff, CA, UNITED STATES
Erickson, Mary Ruth, San Diego, CA, UNITED STATES
Yen, Frances, San Diego, CA, UNITED STATES
Bihain, Bernard, Encinitas, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002037849	A1	20020328
APPLICATION INFO.:	US 2001-776976	A1	20010205 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-758055, filed on 10 Jan 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-176228P	20000114 (60)
	US 2000-198087P	20000413 (60)
	US 2001-299881P	20010621 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 5926
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, JICST-EPLUS, JAPIO, BIOSIS, BIOBUSINESS' ENTERED AT 18:24:55 ON 25 APR 2003

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L1        80414 S SOMATOSTATIN
L2         4567 S L1 AND AGONIST
L3         1034 S BODY WEIGHT REDUCTION
L4          181 S L3 AND METHOD
L5          621 S REDUCE BODY WEIGHT
L6            5 S L5 AND L4

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L7           11 L2 AND L5

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L7 ANSWER 1 OF 11 USPATFULL
TI Combination therapy comprising glucose reabsorption inhibitors and retinoid-X receptor modulators
AB Combination therapy comprising RXR modulators and glucose reabsorption inhibitors useful for the treatment of diabetes and Syndrome X are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:79163 USPATFULL
TITLE: Combination therapy comprising glucose reabsorption inhibitors and retinoid-X receptor modulators
INVENTOR(S): Bussolari, Jacqueline C., Skillman, NJ, UNITED STATES
Chen, Xiaoli, Belle Mead, NJ, UNITED STATES
Conway, Bruce R., Doylestown, PA, UNITED STATES
Demarest, Keith T., Flemington, NJ, UNITED STATES
Ross, Hamish N.M., Far Hills, NJ, UNITED STATES
Severino, Rafael, Madrid, SPAIN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003055091	A1	20030320
APPLICATION INFO.:	US 2002-115725	A1	20020403 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-281479P	20010404 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003
NUMBER OF CLAIMS: 79
EXEMPLARY CLAIM: 1
LINE COUNT: 2308
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 2 OF 11 USPATFULL
TI Combination therapy comprising glucose reabsorption inhibitors and PPAR modulators
AB Combination therapy comprising PPAR modulators and glucose reabsorption inhibitors useful for the treatment of diabetes and Syndrome X are disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:65429 USPATFULL
TITLE: Combination therapy comprising glucose reabsorption inhibitors and PPAR modulators
INVENTOR(S): Bussolari, Jacqueline C., Skillman, NJ, UNITED STATES
Chen, Xiaoli, Belle Mead, NJ, UNITED STATES
Conway, Bruce R., Doylestown, PA, UNITED STATES
Demarest, Keith T., Flemington, NJ, UNITED STATES
Ross, Hamish N.M., Far Hills, NJ, UNITED STATES
Severino, Rafael, Madrid, SPAIN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003045553	A1	20030306
APPLICATION INFO.:	US 2002-115827	A1	20020403 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-281429P	20010404 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003
NUMBER OF CLAIMS: 67
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 2106
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 3 OF 11 USPATFULL
TI GPR10 as a target for identifying weight modulating compounds
AB The invention features assays for the identification of compounds useful for the modulation of body weight. Such compounds are useful for the treatment of obesity and cachexia. The methods of the invention involve cell-free and cell-based assays that identify compounds which bind to and/or activate or inhibit the activity of GPR10, a G protein-coupled receptor, followed by an in vivo assay of the effect of the compound on feeding behavior, body weight, or metabolic rate. The invention also features compounds which bind to and/or activate or inhibit the activity

of GPR10 as well as pharmaceutical compositions comprising such compounds.

In addition, the invention includes nucleic acid molecules comprising a nucleotide sequence encoding all or a portion of murine GPR10, polypeptides comprising all or a portion of murine GPR10, antibodies directed against murine GPR10, and animals harboring a murine GPR10 transgene (e.g., mice overexpressing murine GPR10).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:123413 USPATFULL
TITLE:	GPR10 as a target for identifying weight modulating compounds
INVENTOR(S):	Stricker-Kongra, Alain, Watertown, MA, United States Gu, Wei, Brookline, MA, United States
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001010921	A1	20010802
	US 6537765	B2	20030325
APPLICATION INFO.:	US 2001-799955	A1	20010306 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1998-172353, filed on 14 Oct 1998, GRANTED, Pat. No. US 6197530		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-101380P	19980922 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	ANITA L. MEIKLEJOHN, PH.D., Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS:	19
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	9 Drawing Page(s)
LINE COUNT:	1987

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 4 OF 11 USPATFULL

TI GPR10 as a target for identifying weight modulating compounds

AB The invention features assays for the identification of compounds useful for the modulation of body weight. Such compounds are useful for the treatment of obesity and cachexia. The methods of the invention involve cell-free and cell-based assays that identify compounds which bind to and/or activate or inhibit the activity of GPR10, a G protein-coupled receptor, followed by an in vivo assay of the effect of the compound on feeding behavior, body weight, or metabolic rate. The invention also features compounds which bind to and/or activate or inhibit the activity of GPR10 as well as pharmaceutical compositions comprising such compounds. In addition, the invention includes nucleic acid molecules comprising a nucleotide sequence encoding all or a portion of murine GPR10, polypeptides comprising all or a portion of murine GPR10, antibodies directed against murine GPR10, and animals harboring a murine GPR10 transgene (e.g., mice overexpressing murine GPR10).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:33027 USPATFULL
TITLE:	GPR10 as a target for identifying weight modulating compounds
INVENTOR(S):	Stricker-Krongrad, Alain, Lexington, MA, United States Gu, Wei, Brookline, MA, United States
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6197530	B1	20010306
APPLICATION INFO.:	US 1998-172353		19981014 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-101380P	19980922 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Mertz, Prema
ASSISTANT EXAMINER: Hamud, Fozia
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1990
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 5 OF 11 USPATFULL
TI Modulators of ob gene and screening methods therefor
AB This invention relates to the isolation and cloning of the promoter and other control regions of a human ob gene. It provides a method for identifying and screening for agents useful for the treatment of diseases and pathological conditions affected by the level of expression of an ob gene. These agents interact directly or indirectly with the promoter or other control regions of the ob gene. A PPAR.gamma. **agonist**, BRL49653, has been identified to be useful in treating anorexia, cachexia, and other diseases characterized by insufficient food intake or body weight loss. Modulators of ob gene expression may be used to treat other diseases such as obesity, diabetes, hypertension, cardiovascular diseases and infertility.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:67567 USPATFULL
TITLE: Modulators of ob gene and screening methods therefor
INVENTOR(S): Briggs, Michael R., Downingtown, PA, United States
Auwerx, Johan, Millionfosse, France
de Vos, Piet, Zingem, Belgium
Staels, Bart, Kraainem, Belgium
Croston, Glenn E., San Diego, CA, United States
Miller, Stephen G., San Diego, CA, United States
PATENT ASSIGNEE(S): Ligand Pharmaceuticals Incorporated, San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6068976		20000530
APPLICATION INFO.:	US 1996-618100		19960319 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-558588, filed on 30 Oct 1995, now abandoned which is a continuation-in-part of Ser. No. US 1995-510584, filed on 2 Aug 1995, now abandoned which is a continuation-in-part of Ser. No. US 1995-418096, filed on 5 Apr 1995, now abandoned which is a continuation-in-part of Ser. No. US 1995-408584, filed on 20 Mar 1995, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Yucel, Remy
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Figure(s); 21 Drawing Page(s)

LINE COUNT: 3662
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 6 OF 11 USPATFULL
TI Pharmaceutical compositions containing antibodies to amylin
AB Compositions comprising antibodies directed to amylin in a pharmaceutically acceptable carrier for use in blocking the effects of amylin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1999:99375 USPATFULL		
TITLE:	Pharmaceutical compositions containing antibodies to amylin		
INVENTOR(S):	Cooper, Garth J.S., Auckland, New Zealand Greene, Jr., Howard, Rancho Santa Fe, CA, United States		
PATENT ASSIGNEE(S):	Amylin Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5942227		19990824
APPLICATION INFO.:	US 1996-584578		19960111 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1994-316199, filed on 30 Sep 1994, now abandoned which is a continuation of Ser. No. US 1992-901339, filed on 19 Jun 1992, now abandoned which is a division of Ser. No. US 1991-715302, filed on 4 Jun 1991, now patented, Pat. No. US 5266561 which is a continuation of Ser. No. US 1988-275475, filed on 23 Nov 1988, now abandoned which is a continuation-in-part of Ser. No. US 1988-142447, filed on 11 Jan 1988, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Huff, Sheela		
LEGAL REPRESENTATIVE:	Lyon & Lyon LLP		
NUMBER OF CLAIMS:	2		
EXEMPLARY CLAIM:	1		
LINE COUNT:	1193		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 7 OF 11 USPATFULL
TI Treatment of type 2 diabetes mellitus
AB Antibody methods for blocking the effects of diabetes-associated peptide, or "amylin", a hormone found in the amyloid masses of Type 2 diabetics, are disclosed. This putative hormone has been discovered to function both to inhibit insulin secretion and to inhibit glycogen synthesis. Regulation is accomplished by blocking the binding of amylin or amylin agonists, including calcitonin gene related peptide (CGRP), or biologically active sub-peptides thereof. Inhibitors include antibodies directed to amylin and amylin **agonist** active sites. Other antagonists include anti-idiotype antibodies directed to antibodies directed to amylin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1998:14479 USPATFULL		
TITLE:	Treatment of type 2 diabetes mellitus		
INVENTOR(S):	Cooper, Garth J.S., Woodstock, England Greene, Jr., Howard, Rancho Santa Fe, CA, United States		
PATENT ASSIGNEE(S):	Amylin Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5716619		19980210

APPLICATION INFO.: US 1994-295361 19940823 (8)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1992-901338, filed on 19 Jun 1992, now abandoned which is a continuation of Ser. No. US 1991-715302, filed on 4 Jun 1991, now patented, Pat. No. US 5266561 which is a continuation of Ser. No. US 1988-275475, filed on 23 Nov 1988, now abandoned which is a continuation-in-part of Ser. No. US 1988-142447, filed on 11 Jan 1988, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hutzell, Paula K.
ASSISTANT EXAMINER: Prickril, Benet
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
LINE COUNT: 1181
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 8 OF 11 USPATFULL
TI Treatment of insulin resistance
AB Compounds and methods for blocking the effects of diabetes-associated peptide, or "amylin", a hormone found in the amyloid masses of Type 2 diabetics. This putative hormone has been discovered to function both to inhibit insulin secretion and to inhibit glycogen synthesis. Regulation is accomplished by blocking the binding of amylin or amylin agonists, including calcitonin gene related peptide (CGRP], or biologically active sub-peptides thereof. Inhibitors include substituted peptides or sub-peptides of amylin or CGRP, cross-linked amylin and amylin agonists, synthetic amylin, anti-amylin receptor antibodies and anti-idiotype antibodies, and antibodies directed to amylin and amylin **agonist** active sites. Other antagonists include organic compounds which can be screened and assayed for anti-amylin effects by disclosed methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 94:7674 USPATFULL
TITLE: Treatment of insulin resistance
INVENTOR(S): Cooper, Garth J. S., Woodstock, England
Greene, Jr., Howard, Rancho Sante Fe, CA, United States
PATENT ASSIGNEE(S): Amylin Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5281581		19940125
APPLICATION INFO.:	US 1992-901602		19920619 (7)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1991-715302, filed on 4 Jun 1991 which is a continuation of Ser. No. US 1988-275475, filed on 23 Nov 1988, now abandoned which is a continuation-in-part of Ser. No. US 1988-142447, filed on 11 Jan 1988, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Lee, Lester L.
LEGAL REPRESENTATIVE: Lyon & Lyon
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
LINE COUNT: 1162
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 9 OF 11 USPATFULL
TI Treatment of type 2 diabetes mellitus
AB Compounds and methods for blocking the effects of diabetes-associated peptide, or "amylin", a hormone found in the amyloid masses of Type 2 diabetics. This putative hormone has been discovered to function both to

inhibit insulin secretion and to inhibit glycogen synthesis. Regulation is accomplished by blocking the binding of amylin or amylin agonists, including calcitonin gene related peptide (CGRP], or biologically active sub-peptides thereof. Inhibitors include substituted peptides or sub-peptides of amylin or CGRP, cross-linked amylin and amylin agonists, synthetic amylin, anti-amylin receptor antibodies and anti-idiotype antibodies, and antibodies directed to amylin and amylin **agonist** active sites. Other antagonists include organic compounds which can be screened and assayed for anti-amylin effects by disclosed methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	93:100737 USPATFULL
TITLE:	Treatment of type 2 diabetes mellitus
INVENTOR(S):	Cooper, Garth J. S., Woodstock, United Kingdom Greene, Jr., Howard, Rancho Santa Fe, CA, United States
PATENT ASSIGNEE(S):	Amylin Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5266561		19931130
APPLICATION INFO.:	US 1991-715302		19910604 (7)

RELATED APPLN. INFO.:	Continuation of Ser. No. US 1988-275475, filed on 23 Nov 1988, now abandoned which is a continuation-in-part of Ser. No. US 1988-142447, filed on 11 Jan 1988, now abandoned
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Lee, Lester L.
LEGAL REPRESENTATIVE:	Lyon & Lyon
NUMBER OF CLAIMS:	4
EXEMPLARY CLAIM:	1
LINE COUNT:	1180

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 10 OF 11 USPATFULL

TI Hypoglycemics

AB Non-insulin dependent, or type 2, diabetes mellitus in a patient is treated by administering to the patient a hypoglycemic agent that enhances plasma concentrations of amylin and a therapeutically effective amount of an amylin antagonist. Hypoglycemic agents which enhance plasma concentrations of amylin can be sulfonylureas such as glibenclamide and tolbutamide. Amylin antagonists can be amylin 8-37 and CGRP 8-37. Administration of the amylin antagonist in conjunction with the hypoglycemic agent also enhances the blood glucose lowering effects of the hypoglycemic agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	93:93765 USPATFULL
TITLE:	Hypoglycemics
INVENTOR(S):	Cooper, Garth J. S., Solana Beach, CA, United States Moore, Candace X., San Diego, CA, United States
PATENT ASSIGNEE(S):	Amylin Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5260275		19931109
APPLICATION INFO.:	US 1990-567919		19900814 (7)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Russel, Jeffrey E.
LEGAL REPRESENTATIVE:	Lyon & Lyon
NUMBER OF CLAIMS:	13

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 1883
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 11 OF 11 WPIDS (C) 2003 THOMSON DERWENT
TI Reducing body weight by administration of **somatostatin** or its **agonist** - for treating obese patients or non-insulin-dependent diabetics.
AN 1999-059685 [05] WPIDS
AB WO 9851331 A UPAB: 19990203
Body weight is decreased by administration of **somatostatin** (I) or its agonists (II).
USE - The method is especially used to treat obese subjects or patients with non-insulin dependent diabetes, for therapeutic or cosmetic reasons, both humans and animals.
Dwg.0/0
ACCESSION NUMBER: 1999-059685 [05] WPIDS
DOC. NO. CPI: C1999-017522
TITLE: Reducing body weight by administration of **somatostatin** or its **agonist** - for treating obese patients or non-insulin-dependent diabetics.
DERWENT CLASS: B04
INVENTOR(S): CAWTHORNE, M A; LIU, Y; SENNITT, M V
PATENT ASSIGNEE(S): (SCRC) SCRAS SOC CONSEILS RECH & APPL SCI
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9851331	A1	19981119	(199905)*	EN	40
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9876550	A	19981208	(199916)		
EP 981363	A1	20000301	(200016)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9851331	A1	WO 1998-EP2999	19980513
AU 9876550	A	AU 1998-76550	19980513
EP 981363	A1	EP 1998-924317	19980513
		WO 1998-EP2999	19980513

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9876550	A	Based on	WO 9851331
EP 981363	A1	Based on	WO 9851331

PRIORITY APPLN. INFO: US 1997-854941 19970513

=> d his

(FILE 'HOME' ENTERED AT 18:24:15 ON 25 APR 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, JICST-EPLUS, JAPIO, BIOSIS, BIOBUSINESS' ENTERED AT 18:24:55 ON 25 APR 2003

L1 80414 S SOMATOSTATIN
L2 4567 S L1 AND AGONIST
L3 1034 S BODY WEIGHT REDUCTION
L4 181 S L3 AND METHOD
L5 621 S REDUCE BODY WEIGHT
L6 5 S L5 AND L4
L7 11 S L2 AND L5

=> s l2 and l4
L8 3 L2 AND L4

=> d l8 ti abs ibib tot

L8 ANSWER 1 OF 3 USPATFULL
TI Neo-tryptophan
AB The invention provides a novel amino acid, neo-tryptophan, as well as polypeptides containing this novel amino acid such as neurotensin analogs. In addition, the invention provides neo-tryptophan derivatives, serotonin-like neo-tryptophan derivatives, and polypeptides containing such derivatives. The invention also provides methods for making neo-tryptophan, neo-tryptophan derivatives, serotonin-like neo-tryptophan derivatives, and compositions containing these compounds. Further, the invention provides methods for inducing a neurotensin response in a mammal as well as methods for treating a mammal having a serotonin recognition molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:171124 USPATFULL
TITLE:	Neo-tryptophan
INVENTOR(S):	Richelson, Elliott, Ponte Vedra Beach, FL, United States Cusack, Bernadette Marie, Jacksonville, FL, United States Pang, Yuan-Ping, Rochester, MN, United States McCormick, Daniel J., Rochester, MN, United States Fauq, Abdul, Jacksonville, FL, United States Tyler, Beth Marie, Neptune Beach, FL, United States Boules, Mona, Jacksonville, FL, United States
PATENT ASSIGNEE(S):	Mayo Foundation for Medical Education and Research Minnesota corporation (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001027174	A1	20011004
APPLICATION INFO.:	US 2001-755638	A1	20010105 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1999-289693, filed on 9 Apr 1999, GRANTED, Pat. No. US 6214790		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-81356P	19980410 (60)
	US 1998-92195P	19980709 (60)
	US 1998-98119P	19980827 (60)
	US 1998-112137P	19981214 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	MARK S. ELLINGER, PH.D., Fish & Richardson P.C., P.A., Suite 3300, 60 South Sixth Street, Minneapolis, MN, 55402
NUMBER OF CLAIMS:	57
EXEMPLARY CLAIM:	1

NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 1828
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 2 OF 3 USPATFULL
TI Neo-tryptophan
AB The invention provides a novel amino acid, neo-tryptophan, as well as polypeptides containing this novel amino acid such as neurotensin analogs. In addition, the invention provides neo-tryptophan derivatives, serotonin-like neo-tryptophan derivatives, and polypeptides containing such derivatives. The invention also provides methods for making neo-tryptophan, neo-tryptophan derivatives, serotonin-like neo-tryptophan derivatives, and compositions containing these compounds. Further, the invention provides methods for inducing a neurotensin response in a mammal as well as methods for treating a mammal having a serotonin recognition molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2001:52014 USPATFULL
TITLE: Neo-tryptophan
INVENTOR(S): Richelson, Elliott, Ponte Vedra Beach, FL, United States
Cusack, Bernadette Marie, Jacksonville, FL, United States
Pang, Yuan-Ping, Rochester, MN, United States
McCormick, Daniel J., Rochester, MN, United States
Fauq, Abdul, Jacksonville, FL, United States
Tyler, Beth Marie, Neptune Beach, FL, United States
Boules, Mona, Jacksonville, FL, United States
PATENT ASSIGNEE(S): Mayo Foundation for Medical Education And Research, Rochester, MN, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6214790	B1	20010410
APPLICATION INFO.:	US 1999-289693		19990409 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-81356P	19980410 (60)
	US 1998-92195P	19980709 (60)
	US 1998-98119P	19980827 (60)
	US 1998-112137P	19981214 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Russel, Jeffrey E.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.; P.A.
NUMBER OF CLAIMS: 66
EXEMPLARY CLAIM: 1,16
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1950
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 3 OF 3 USPATFULL
TI Use of GLP-1 analogs and derivatives administered peripherally in regulation of obesity
AB This invention relates the use of glucagon-like peptides such as GLP-1, a GLP-1 analog, or a GLP-1 derivative in methods and compositions for reducing body weight.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2001:25867 USPATFULL
TITLE: Use of GLP-1 analogs and derivatives administered peripherally in regulation of obesity

INVENTOR(S):	DiMarchi, Richard D., Carmel, IN, United States Efendic, Suad, Lidingo, Sweden			
PATENT ASSIGNEE(S):	Eli Lilly and Company, Indianapolis, IN, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6191102	B1	20010220	
APPLICATION INFO.:	US 1997-961405		19971030	(8)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 1996-30213P		19961105	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Saoud, Christine			
LEGAL REPRESENTATIVE:	Davis, Steven G., Maciak, Ronald S.			
NUMBER OF CLAIMS:	20			
EXEMPLARY CLAIM:	1			
LINE COUNT:	1204			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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NEWS 22	Feb 24	PCTGEN now available on STN
NEWS 23	Feb 24	TEMA now available on STN
NEWS 24	Feb 26	NTIS now allows simultaneous left and right truncation
NEWS 25	Feb 26	PCTFULL now contains images
NEWS 26	Mar 04	SDI PACKAGE for monthly delivery of multifile SDI results
NEWS 27	Mar 19	APOLLIT offering free connect time in April 2003
NEWS 28	Mar 20	EVENTLINE will be removed from STN
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NEWS 30	Mar 24	Additional information for trade-named substances without structures available in REGISTRY
NEWS 31	Apr 11	Display formats in DGENE enhanced
NEWS 32	Apr 14	MEDLINE Reload
NEWS 33	Apr 17	Polymer searching in REGISTRY enhanced
NEWS 34	Apr 21	Indexing from 1947 to 1956 being added to records in CA/CAPLUS
NEWS 35	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX

NEWS EXPRESS	April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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=> s somatostatin
L1 80414 SOMATOSTATIN

=> s l1 and agonist
L2 4567 L1 AND AGONIST

=> s body weight reduction
L3 1034 BODY WEIGHT REDUCTION

=> s l3 and method

L4 181 L3 AND METHOD

=> s reduce body weight
L5 621 REDUCE BODY WEIGHT

=> s l5 and l4
L6 5 L5 AND L4

=> d l5 ti abs ibib tot

L5 ANSWER 1 OF 621 MEDLINE
TI Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial.
AB CONTEXT: Obesity is an independent risk factor for cardiovascular disease, which may be mediated by increased secretion of proinflammatory cytokines by adipose tissue. OBJECTIVE: To determine the effect of a program of changes in lifestyle designed to obtain a sustained reduction of body weight on markers of systemic vascular inflammation and insulin resistance. DESIGN AND SETTING: Randomized single-blind trial conducted from February 1999 to February 2002 at a university hospital in Italy. PATIENTS: One hundred twenty premenopausal obese women (body mass index >/=30) aged 20 to 46 years without diabetes, hypertension, or hyperlipidemia. INTERVENTIONS: The 60 women randomly assigned to the intervention group received detailed advice about how to achieve a reduction of weight of 10% or more through a low-energy Mediterranean-style diet and increased physical activity. The control group (n = 60) was given general information about healthy food choices and exercise. MAIN OUTCOME MEASURES: Lipid and glucose intake; blood pressure; homeostatic model assessment of insulin sensitivity; and circulating levels of interleukin 6 (IL-6), interleukin 18 (IL-18), C-reactive protein (CRP), and adiponectin. RESULTS: After 2 years, women in the intervention group consumed more foods rich in complex carbohydrates (9% corrected difference; P<.001), monounsaturated fat (2%; P =.009), and fiber (7 g/d; P<.001); had a lower ratio of omega-6 to omega-3 fatty acids (-5; P<.001); and had lower energy (-310 kcal/d; P<.001), saturated fat (-3.5%; P =.007), and cholesterol intake (-92 mg/d; P<.001) than controls. Body mass index decreased more in the intervention group than in controls (-4.2; P<.001), as did serum concentrations of IL-6 (-1.1 pg/mL; P =.009), IL-18 (-57 pg/mL; P =.02), and CRP (-1.6 mg/L; P =.008), while adiponectin levels increased significantly (2.2 micro g/mL; P =.01). In multivariate analyses, changes in free fatty acids (P =.008), IL-6 (P =.02), and adiponectin (P =.007) levels were independently associated with changes in insulin sensitivity. CONCLUSION: In this study, a multidisciplinary program aimed to **reduce body weight** in obese women through lifestyle changes was associated with a reduction in markers of vascular inflammation and insulin resistance.

ACCESSION NUMBER: 2003167016 IN-PROCESS
DOCUMENT NUMBER: 22571306 PubMed ID: 12684358
TITLE: Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial.
AUTHOR: Esposito Katherine; Pontillo Alessandro; Di Palo Carmen; Giugliano Giovanni; Masella Mariangela; Marfella Raffaele; Giugliano Dario
CORPORATE SOURCE: Center for Obesity Management, Department of Geriatrics and Metabolic Diseases, Chair of Plastic and Reconstructive Surgery, Department of Psychiatry, and Cardiovascular Research Center, Second University of Naples, Naples, Italy.
SOURCE: JAMA, (2003 Apr 9) 289 (14) 1799-804.
Journal code: 7501160. ISSN: 0098-7484.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals
ENTRY DATE: Entered STN: 20030410
Last Updated on STN: 20030410

L5 ANSWER 2 OF 621 MEDLINE
TI 5-HT(2C) Receptor Agonists as Potential Drugs for the Treatment of Obesity.
AB An association between the brain serotonin (5-HT) system and feeding has been postulated since the 1970's but it has only been in recent years that the nature of 5-HT-mediated hypophagia has become well understood, and the receptor subtypes responsible for the effect better defined. The invention and utilisation of subtype-selective 5-HT receptor antagonists has demonstrated that the 5-HT(2C) receptor is of paramount importance in this regard. Importantly, ethological studies of animal behaviour have shown that the hypophagia resulting from 5-HT(2C) receptor activation is likely to be a consequence of increased satiety and this is in contrast to hypophagia following 5-HT(2C) receptor activation. Furthermore, recent studies have also shown that 5-HT(2C) receptor agonists not only reduce feeding when acutely administered to rats or mice, they can also **reduce body weight** without inducing tolerance when administered chronically to obese animals. These observations have led researchers to conclude that selective 5-HT(2C) receptor agonists have the potential to be effective anti-obesity agents. Encouragingly, this suggestion is supported by both direct and indirect evidence from clinical studies. Indirect evidence stems from recent observations that the clinically effective anorectic agent d-fenfluramine exerts its hypophagic and weight-loss effects via 5-HT(2C) receptor activation. More direct clinical evidence derives from the use of the prototypical 5-HT(2C) receptor agonist m-chlorophenylpiperazine (mCPP), with which both acute hypophagia and body-weight loss have been observed. The current paper therefore reviews both the pre-clinical and clinical evidence supporting the use of 5-HT(2C) receptor agonists for the treatment of obesity and assesses the developments that have been made in this regard to date.
ACCESSION NUMBER: 2003162048 IN-PROCESS
DOCUMENT NUMBER: 22565773 PubMed ID: 12678838
TITLE: 5-HT(2C) Receptor Agonists as Potential Drugs for the Treatment of Obesity.
AUTHOR: Bickerdike Michael J
CORPORATE SOURCE: Department of Molecular Pharmacology, Vernalis Research Ltd., Oakdene Court, 613 Reading Road. Winnersh, Wokingham, RG41 5UA, UK; E-mail:. M.Bickerdike@vernalis.com
SOURCE: Curr Top Med Chem, (2003) 3 (8) 885-97.
Journal code: 101119673. ISSN: 1568-0266.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20030408
Last Updated on STN: 20030408

L5 ANSWER 3 OF 621 MEDLINE
TI The importance of a high feed intake during lactation of primiparous sows nursing large litters.
AB The objective of this study was to investigate whether nursing a large number of piglets has negative effects on lactation and postweaning performance of primiparous sows and whether a greater lactation feed intake can prevent possible negative effects. Data were recorded on 268 ad libitum-fed sows of three genotypes (G1, G2, and G3) in an experiment where litter size was standardized to 8, 11, or 14 piglets during a 4-wk lactation. Compared to G1 and G2, G3 sows were heavier ($P < 0.05$) and leaner ($P < 0.05$) at weaning of their litters, lost similar amounts of BW and backfat, and their piglets grew faster ($P < 0.05$). Compared to G1, feed intake during lactation was higher for G3 sows ($P < 0.05$), and their

risk of a prolonged weaning-to-estrus interval was lower (P < 0.01). Daily feed intake by sows was not affected by litter size in G1 and G3, but it was quadratically affected in G2 (P < 0.05), with a maximum at 10.8 piglets. Backfat loss of the sows increased linearly with litter size (P < 0.05) in G1 and G3. In G2, backfat loss increased only at litter sizes > 9.8 piglets (P < 0.01). Body weight loss of the sow and litter weight gain increased linearly with litter size (P < 0.001). Per extra piglet nursed, sows had a 23% (P < 0.01) higher probability of a prolonged weaning-to-estrus interval. A higher daily feed intake during lactation reduced tissue loss of the sow, increased litter weight gain (P < 0.01), and reduced the probability of a prolonged weaning-to-estrus interval (by 42% per extra kilogram; P < 0.01). Sows with a lower daily body weight loss during first lactation had a larger second litter (1.28 piglets/kg; P < 0.01), and their probability of a prolonged weaning-to-estrus interval was reduced by 61% per kilogram (P < 0.001). With increasing litter size, it is therefore recommended to **reduce body weight** loss during lactation by stimulating daily feed intake and by genetic selection.

ACCESSION NUMBER: 2003145847 IN-PROCESS
DOCUMENT NUMBER: 22547803 PubMed ID: 12661638
TITLE: The importance of a high feed intake during lactation of primiparous sows nursing large litters.
AUTHOR: Eissen J J; Apeldoorn E J; Kanis E; Verstegen M W A; de Greef K H
CORPORATE SOURCE: Animal Breeding and Genetics Group, Wageningen Institute of Animal Sciences, Wageningen University, 6700 AH Wageningen, The Netherlands.
SOURCE: JOURNAL OF ANIMAL SCIENCE, (2003 Mar) 81 (3) 594-603. Journal code: 8003002. ISSN: 0021-8812.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20030331
Last Updated on STN: 20030331

L5 ANSWER 4 OF 621 MEDLINE
TI A soybean peptide isolate diet promotes postprandial carbohydrate oxidation and energy expenditure in type II diabetic mice.
AB The aim of the present study was to determine the effects of dietary proteins on the oxidation of dietary carbohydrate and lipids in type II diabetic mice. KK-A(y) strain mice were provided free access to a high fat diet (30% of energy as fat) for an initial 4-wk period to induce diabetes. To **reduce body weight** gain, the mice were subsequently fed restrictive isoenergetic and isonitrogenous diets (35% of energy as protein and 5% as fat) based on either casein or soy protein isolate hydrolysate (SPI-H) for 4 wk. To measure exogenous carbohydrate and lipid oxidation, the mice were fed a diet containing (13)C-glucose or (13)C-triolein while they were in a respiratory chamber for 72 h. Postprandial energy expenditure was higher in the SPI-H than in the casein group; this difference was due to an increase in postprandial exogenous and endogenous carbohydrate oxidation. There were no differences in 24-h energy expenditure between dietary groups. Oxidation of exogenous carbohydrate tended to be higher (P = 0.054) in the SPI-H group during the 24 h of measurement. Fecal excretion of (13)C-glucose was lower but the excretion of lipid was higher in mice fed the SPI-H diet than in casein-fed mice. These results indicate that in type II diabetic mice, dietary SPI-H not only inhibits the absorption of dietary lipids and increases the absorption of dietary carbohydrates but also augments postprandial energy expenditure, which is accompanied by a postprandial increase in oxidation of dietary carbohydrates.

ACCESSION NUMBER: 2003145573 MEDLINE
DOCUMENT NUMBER: 22499798 PubMed ID: 12612148
TITLE: A soybean peptide isolate diet promotes postprandial

carbohydrate oxidation and energy expenditure in type II diabetic mice.

AUTHOR: Ishihara Kengo; Oyaizu Shinichi; Fukuchi Yoshiko; Mizunoya Wataru; Segawa Kikumi; Takahashi Miki; Mita Yukiko; Fukuya Yoko; Fushiki Tohru; Yasumoto Kyoden

CORPORATE SOURCE: Department of Food and Nutrition, School of Life Studies, Sugiyama Jogakuen University, Nagoya 464-8662, Japan.. kengo@food.sugiyama-u.ac.jp

SOURCE: JOURNAL OF NUTRITION, (2003 Mar) 133 (3) 752-7. Journal code: 0404243. ISSN: 0022-3166.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200304

ENTRY DATE: Entered STN: 20030331
Last Updated on STN: 20030416
Entered Medline: 20030411

L5 ANSWER 5 OF 621 MEDLINE

TI Sub-clinical eating disorder characteristics among male and female triathletes.

AB The characteristics of sub-clinical eating disorders were assessed in 583 male and female triathletes. We found that 28% of the females and 11% of the males scored below the mid-point of the range for the Eating Attitude Test-26 (EAT-26) construct Preoccupation with Food and Weight, and respectively 39% and 23% scored below the mid-point of the range for the construct Calorie Control. All of the subjects indicated dissatisfaction with their actual body mass index (BMI). The study participants revealed attempts to **reduce body weight** by means of energy restriction, severe limitation of food groups and excessive exercise, in addition to controlling their food intake on the basis of strict dietary rules. The triathlon seems to be a sport that is susceptible to a higher prevalence of disordered eating. Further studies are needed to investigate its real prevalence and the factors contributing to it.

ACCESSION NUMBER: 2002690903 MEDLINE

DOCUMENT NUMBER: 22339410 PubMed ID: 12452253

TITLE: Sub-clinical eating disorder characteristics among male and female triathletes.

AUTHOR: DiGioacchino DeBate R; Wethington H; Sargent R

CORPORATE SOURCE: Department of Health Promotion and Kinesiology, University of North Carolina, Charlotte, NC 28223-0001, USA.

SOURCE: EATING AND WEIGHT DISORDERS, (2002 Sep) 7 (3) 210-20. Journal code: 9707113. ISSN: 1124-4909.

PUB. COUNTRY: Italy

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200303

ENTRY DATE: Entered STN: 20021214
Last Updated on STN: 20030322
Entered Medline: 20030321

L5 ANSWER 6 OF 621 MEDLINE

TI Beneficial role of dietary phytoestrogens in obesity and diabetes.

AB Evidence is emerging that dietary phytoestrogens play a beneficial role in obesity and diabetes. Nutritional intervention studies performed in animals and humans suggest that the ingestion of soy protein associated with isoflavones and flaxseed rich in lignans improves glucose control and insulin resistance. In animal models of obesity and diabetes, soy protein has been shown to reduce serum insulin and insulin resistance. In studies of human subjects with or without diabetes, soy protein also appears to moderate hyperglycemia and **reduce body weight**

, hyperlipidemia, and hyperinsulinemia, supporting its beneficial effects on obesity and diabetes. However, most of these clinical trials were relatively short and involved a small number of patients. Furthermore, it is not clear whether the beneficial effects of soy protein and flaxseed are due to isoflavones (daidzein and genistein), lignans (matairesinol and secoisolariciresinol), or some other component. Isoflavones and lignans appear to act through various mechanisms that modulate pancreatic insulin secretion or through antioxidative actions. They may also act via estrogen receptor-mediated mechanisms. Some of these actions have been shown in vitro, but the relevance of these studies to in vivo disease is not known. The diversity of cellular actions of isoflavones and lignans supports their possible beneficial effects on various chronic diseases. Further investigations are needed to evaluate the long-term effects of phytoestrogens on obesity and diabetes mellitus and their associated possible complications.

ACCESSION NUMBER: 2002689618 MEDLINE
DOCUMENT NUMBER: 22338109 PubMed ID: 12450882
TITLE: Beneficial role of dietary phytoestrogens in obesity and diabetes.
AUTHOR: Bhathena Sam J; Velasquez Manuel T
CORPORATE SOURCE: Phytonutrients Laboratory, Beltsville Human Nutrition Research Center, Agricultural Research Service, US Department of Agriculture, Beltsville, MD 20705, USA.. bhathens@ba.ars.usda.gov
SOURCE: AMERICAN JOURNAL OF CLINICAL NUTRITION, (2002 Dec) 76 (6) 1191-201. Ref: 115
Journal code: 0376027. ISSN: 0002-9165.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20021214
Last Updated on STN: 20021221
Entered Medline: 20021220

L5 ANSWER 7 OF 621 MEDLINE
TI Clinical, physiopathological and dietetic aspects of metabolic syndrome.
AB Hypertriglyceridaemia, diabetes, hypertension and obesity are the deadly quartet indicating a syndrome at high risk for cardiovascular disease for which, in 1998, WHO proposed the definition of Metabolic Syndrome, related to an elevated degree of insulin resistance. Treatment will often include behavioural changes that **reduce body weight** and increase physical activity A high-carbohydrate/low-fat diet with complex carbohydrates and mainly unsaturated fat is recommended. Replacing refined grain products and potatoes with minimally processed plant-based foods such as whole grains, fruit, and vegetables, and reducing the intake of high glycaemic load beverages may offer a simple strategy for reducing the incidence of coronary heart disease.

ACCESSION NUMBER: 2002648795 MEDLINE
DOCUMENT NUMBER: 22295658 PubMed ID: 12408457
TITLE: Clinical, physiopathological and dietetic aspects of metabolic syndrome.
AUTHOR: Leonetti F; Lacobellis G; Zappaterreno A; Di Mario U
CORPORATE SOURCE: Endocrinology, Department of Clinical Sciences, La Sapienza University, Rome, Italy.. frida.leonetti@tin.it
SOURCE: DIGESTIVE AND LIVER DISEASE, (2002 Sep) 34 Suppl 2 S134-9.
Journal code: 100958385. ISSN: 1590-8658.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20021105
Last Updated on STN: 20030212
Entered Medline: 20030211

L5 ANSWER 8 OF 621 MEDLINE
TI Dietary alpha-linolenic acid-rich diacylglycerols **reduce body weight** gain accompanying the stimulation of intestinal beta-oxidation and related gene expressions in C57BL/KsJ-db/db mice.
AB Dietary fat contributes to the development of obesity. We examined the effect of dietary diacylglycerol (DG), which is a minor component of edible oils, on the development of obesity and expression of genes involved in energy homeostasis in C57BL/KsJ-db/db mice. Mice were fed diets containing either 14 g/100 g (%) triacylglycerol (TG), 10% TG + 4% alpha-linolenic acid-rich TG (ALATG), or 10% TG + 4% alpha-linolenic acid-rich diacylglycerol (ALADG) for 1 mo. Mice fed ALADG, but not ALATG had less body weight gain and higher rectal temperature than the TG-fed controls. These effects were accompanied by up-regulation of acyl-CoA oxidase, medium-chain acyl-CoA dehydrogenase, fatty acid binding protein, and uncoupling protein (UCP)-2 mRNA and beta-oxidation activity in the small intestine. In contrast, the treatments did not affect beta-oxidation and related gene expressions in the liver or UCP-3 mRNA level in skeletal muscle. These results indicate that stimulation of lipid metabolism in the small intestine might be closely related to the antiobesity and thermogenic effects of dietary DG. In addition, structural differences between DG and TG, not variations in the composition of fatty acids, are responsible for the different effects of the lipids.
ACCESSION NUMBER: 2002611231 MEDLINE
DOCUMENT NUMBER: 22255120 PubMed ID: 12368389
TITLE: Dietary alpha-linolenic acid-rich diacylglycerols **reduce body weight** gain accompanying the stimulation of intestinal beta-oxidation and related gene expressions in C57BL/KsJ-db/db mice.
AUTHOR: Murase Takatoshi; Nagasawa Azumi; Suzuki Junko; Wakisaka Takuya; Hase Tadashi; Tokimitsu Ichiro
CORPORATE SOURCE: Biological Science Laboratories, Kao Corporation, Ichikai, Haga, Tochigi 321-3497, Japan.
SOURCE: JOURNAL OF NUTRITION, (2002 Oct) 132 (10) 3018-22. Journal code: 0404243. ISSN: 0022-3166.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 20021008
Last Updated on STN: 20021030
Entered Medline: 20021029

L5 ANSWER 9 OF 621 MEDLINE
TI Diarrhoeal disease control.
AB 1.5 million children under the age of 5 years die of diarrheal disease every year. The incidence of diarrhea is highest among children 3-36 months of age with peak incidence between 6-9 months. Causes of diarrheal disease are poor sanitary facilities, poverty and ignorance leading to malnutrition, overcrowding, and unhygenic living conditions. Significant advances in knowledge have contributed to better treatment and control of diarrheal disease: the use of new viral and bacterial agents have aided in identifying the causative agents in 70% of diarrheal cases. Breastfeeding protects infants from the diseases. Water supply and sanitary conditions help to prevent diarrhea; however, these alone do not sufficiently control acute diarrheal disease. The primary cause of mortality from diarrheal disease is dehydration. In cholera, fluid losses can **reduce**

body weight by 10% in 4-6 hours. Oral rehydration solution (ORS) has recently simplified the procedure of rehydration. Evidence indicates that the use of ORS at the household level can decrease the mortality to below 1%. Effective implementation of ORS at the community level depends on: 1) production of adequate quality of packets of ORS, 2) extensive training of health personnel, 3) education of mothers in the treatment of ORS, and 4) easy availability of ORS packets. In India, the Ministry of Health is giving high priority to large scale production of low priced ORS packets. In addition, training courses for medical and paramedical workers are being organized throughout the country. Control of diarrheal disease involves the implementation of improvements in water supply, sanitation, health education, and personal hygiene.

ACCESSION NUMBER: 2002595493 MEDLINE
DOCUMENT NUMBER: 21807529 PubMed ID: 12264153
TITLE: Diarrhoeal disease control.
AUTHOR: Misra B S
SOURCE: Swasth Hind, (1981 Mar-Apr) 25 (3-4) 92-3.
Journal code: 21040350R. ISSN: 0586-1179.
Report No.: PIP-008031; POP-00108459.
PUB. COUNTRY: India
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Population
ENTRY MONTH: 198207
ENTRY DATE: Entered STN: 20021101
Last Updated on STN: 20021101
Entered Medline: 19820707

L5 ANSWER 10 OF 621 MEDLINE
TI [Differential type 2 diabetes therapy based on pathophysiological aspects].
Differentialtherapie des Typ 2 Diabetes aufgrund pathophysiologischer Aspekte.
AB Type 2 Diabetes is characterized by a deficit in early insulin secretion and increased insulin resistance. Based on the measurement of fasting and 2 h postprandial blood glucose a simple typing of hyperglycemia is possible in isolated fasting hyperglycemia (IFH), isolated postprandial hyperglycemia (IPH) and combined hyperglycemia (CH). IFH is predominantly associated with insulin resistance whereas in IPH a more pronounced insulin deficit is found. This and other simple parameters such as BMI, comorbidities and age are the basis of differential therapy with OAD if best efforts with life style modification fail to reach the target values of the lipid triad. Patients with IFH profit particularly from metformin and long acting sulfonylureas (glimepiride, glibenclamide). Patients with IPH are candidates for prandial antidiabetics (AGI, nateglinide, repaglinide). Antihyperglycemic agents such as metformin and AGI bear no risk of hyperglycemia and **reduce body weight**. Prandial insulin secretagogues have lower risk of weight gain and hypoglycemia than long acting sulfonylureas. They are therefore beneficial in obese patients and the elderly. The same principles are valid for combination treatment. With exception of insulin secretagogues all OAD can be combined if monotherapy fails to reach the target levels of the gluco-triad. Instead of a stepwise treatment algorithm an individualized therapy based on pathophysiology and comorbidities taking into account the global risk seems to be beneficial.

ACCESSION NUMBER: 2002474223 MEDLINE
DOCUMENT NUMBER: 22221581 PubMed ID: 12235731
TITLE: [Differential type 2 diabetes therapy based on pathophysiological aspects].
Differentialtherapie des Typ 2 Diabetes aufgrund pathophysiologischer Aspekte.
AUTHOR: Hanefeld M; Fischer S
CORPORATE SOURCE: Zentrum fur Klinische Studien, GWT-TU Dresden..

hanefeld@gwt-tud.de
SOURCE: THERAPEUTISCHE UMSCHAU, (2002 Aug) 59 (8) 393-401.
Journal code: 0407224. ISSN: 0040-5930.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20020919
Last Updated on STN: 20021217
Entered Medline: 20021204

L5 ANSWER 11 OF 621 MEDLINE
TI [From obesity to diabetes].
Von der Adipositas zum Diabetes.
AB The major risk factor for the development of insulin resistance and type 2 diabetes is obesity. A key role is the new understanding of adipocytes as an endocrine system. Adipocytes secrete numerous substances that contribute to peripheral insulin resistance, including adiponectin, resistin, TNF-alpha and interleukin 6. There is also a role of free fatty acids by blocking directly intracellular metabolism of glucose and by their lipotoxicity. The pre-receptor metabolism of cortisol may be enhanced in visceral adipose tissue by activation of 11 beta-hydroxysteroid dehydrogenase type 1. The new class of thiazolidinediones (glitazones), binding to the peroxisome proliferator activated receptor (PPAR-gamma) lowers the levels of resistin and increases adiponectin, resulting in an improvement of glucose homeostasis. However, the first step to avoid insulin resistance should be an attempt to **reduce body weight** and to increase physical activity. These are successful means to avoid the development of type 2 diabetes from prediabetic states, as shown recently in 3 independent intervention trials.
ACCESSION NUMBER: 2002474222 MEDLINE
DOCUMENT NUMBER: 22221580 PubMed ID: 12235730
TITLE: [From obesity to diabetes].
Von der Adipositas zum Diabetes.
AUTHOR: Stockli R; Keller U
CORPORATE SOURCE: Abteilung fur Endokrinologie, Diabetologie und Klinische Ernahrung, Departement Innere Medizin, Universitatskliniken Kantonsspital, Basel.
SOURCE: THERAPEUTISCHE UMSCHAU, (2002 Aug) 59 (8) 388-92.
Journal code: 0407224. ISSN: 0040-5930.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20020919
Last Updated on STN: 20021217
Entered Medline: 20021204

L5 ANSWER 12 OF 621 MEDLINE
TI Nicotine treatment decreases food intake and body weight via a leptin-independent pathway in Psammomys obesus.
AB It has been reported previously that leptin may be involved in nicotine's ability to **reduce body weight**. Our aim was to investigate whether the anorexic action of nicotine is related to the actions of leptin by utilizing lean leptin-sensitive and obese leptin-resistant Psammomys obesus. Lean and obese P. obesus were assigned to receive nicotine sulphate at 6, 9 or 12 mg/day or saline (control) for 9 days (n = 6-10 in each group), administered using mini-osmotic pumps. Food intake, body weight, plasma leptin concentrations, plasma insulin and blood glucose were measured at baseline and throughout the study period. Nicotine treatment reduced food intake by up to 40% in lean and obese P.

obesus. Plasma leptin levels fell significantly only in lean nicotine-treated animals, whereas no changes were observed in obese nicotine-treated animals. However, both lean and obese nicotine-treated animals had similar reductions in body weight. Our results show that nicotine has dramatic effects on food intake and body weight, however, these changes appear to be independent of the leptin signalling pathway.

ACCESSION NUMBER: 2002457907 MEDLINE
DOCUMENT NUMBER: 22178678 PubMed ID: 12190999
TITLE: Nicotine treatment decreases food intake and body weight via a leptin-independent pathway in Psammomys obesus.
AUTHOR: Sanigorski A; Fahey R; Cameron-Smith D; Collier G R
CORPORATE SOURCE: Metabolic Research Unit, School of Health Sciences, Deakin University, Waurn Ponds, Victoria, Australia.. andrewjs@deakin.edu.au
SOURCE: DIABETES, OBESITY & METABOLISM, (2002 Sep) 4 (5) 346-50. Journal code: 100883645. ISSN: 1462-8902.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 20020910
Last Updated on STN: 20030312
Entered Medline: 20030311

L5 ANSWER 13 OF 621 MEDLINE
TI Daily walking reduces visceral adipose tissue areas and improves insulin resistance in Japanese obese subjects.
AB OBJECTIVE: It is known that the accumulation of abdominal fat is one of the risk factors for atherosclerosis. Although exercise is commonly prescribed to **reduce body weight**, the efficacy of low intensity exercise for the reduction of abdominal visceral adipose tissue remains to be investigated. RESEARCH DESIGN AND METHODS: Thirty one obese Japanese males (body mass index (BMI) > or = 25) ranging in age from 32 to 59, participated in a 1-year follow up study and they were instructed to have a modest increase in daily activity and record their daily walking. Before and after exercise prescription, body composition, blood pressure, physical fitness i.e. aerobic exercise level, muscle strength and flexibility were recorded. Insulin resistance was evaluated using a homeostasis model assessment, the HOMA index. RESULTS: HOMA index, parameters of body composition, blood pressure, triglyceride and HDL cholesterol were significantly improved. The aerobic exercise level, leg strength, weight-bearing index (leg strength/body weight) and the steps taken per day were significantly increased. By stepwise multiple regression analysis, Delta visceral adipose tissue area was the major determinant for Delta HOMA index. (Delta HOMA index=-0.386+0.016 Delta visceral adipose tissue area, r2=0.267, P<0.01). Exercise capacity and calorie intake were not significantly related to Delta visceral adipose tissue area, while Delta steps per day was significantly correlated with Delta visceral adipose tissue area (Delta visceral adipose tissue area=-21.363-0.004 Delta steps per day, r2=0.184, P=0.0326). CONCLUSIONS: Taken together, intra-abdominal visceral adipose tissue is critically involved in insulin resistance and daily walking rather than improvement of exercise capacity correlated with the reduction of visceral adipose tissue in obese Japanese males.

ACCESSION NUMBER: 2002455114 MEDLINE
DOCUMENT NUMBER: 22202053 PubMed ID: 12213351
TITLE: Daily walking reduces visceral adipose tissue areas and improves insulin resistance in Japanese obese subjects.
AUTHOR: Miyatake Nobuyuki; Nishikawa Hidetaka; Morishita Akie; Kunitomi Mie; Wada Jun; Suzuki Hisao; Takahashi Kayo; Makino Hirofumi; Kira Shohei; Fujii Masafumi
CORPORATE SOURCE: Okayama Southern Institute of Health, 408-1 Hirata, 700-0952 Okayama, Japan.. center@okakenko.jp

SOURCE: DIABETES RESEARCH AND CLINICAL PRACTICE, (2002 Nov) 58 (2) 101-7.
Journal code: 8508335. ISSN: 0168-8227.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20020906
Last Updated on STN: 20030424
Entered Medline: 20030423

L5 ANSWER 14 OF 621 MEDLINE

TI Effects of the rat hepatic peroxisome proliferator, Wyeth 14,643, on the lactating goat.

AB Some peroxisome proliferators have been reported to **reduce body weight** gain in suckling rats, possibly through a lactational effect. Decreases in milk production or nutritional quality, either as a result of peroxisome proliferator-induced reductions in lipid content or alterations in the hormonal milieu necessary for milk production, could result in pup growth retardation. Wyeth-14,643 (WY) is hypolipidemic agent and a potent inducer of hepatic peroxisome proliferation in rats and mice. As is commonly seen with rodent hepatic peroxisome proliferators, WY produces minimal or no peroxisome induction in guinea pigs or non-human primates. Goats are an excellent model for studying lactation, however, their sensitivity to peroxisome proliferating chemicals is not known. The present study was performed to assess the sensitivity of goats to the hypolipidemic and peroxisome proliferator properties of WY and to determine the effects of WY on milk quantity and quality. Six lactating adult female goats were assigned to either control or treated groups. Goats in the treated group were administered WY (40 mg/kg/day) for 14 consecutive days. The goats were milked twice daily in order to maintain lactation and the quantity of milk collected was recorded. Milk quality was evaluated by determining the content of total fat, protein, and carbohydrate in milk samples collected following 7 and 14 days of treatment. WY administration had no effects on final body weight, liver weight or, gross and histopathological findings. Milk quantity and quality were unaffected by treatment. Serum cholesterol and triglyceride levels were reduced by 25% compared to controls, although only the difference in cholesterol was statistically significant. Hepatic beta-oxidation (3 x control) and aromatase (1.5 x control) activities were significantly greater in the treatment group; however, there was no treatment-related effect in the total content of hepatic cytochrome P450. There was no difference in aromatase activity in a pooled ovarian microsome sample. Milk estradiol and prolactin concentrations were not affected by treatment. These findings indicate that goats are weak responders to the hepatic peroxisome proliferator effects of WY. Additionally, the slight serum hypolipidemic effect does not impact milk production or nutritional value.

ACCESSION NUMBER: 2002419164 MEDLINE
DOCUMENT NUMBER: 22163583 PubMed ID: 12173247
TITLE: Effects of the rat hepatic peroxisome proliferator, Wyeth 14,643, on the lactating goat.
AUTHOR: Cappon G D; Liu R C M; Frame S R; Hurtt M E
CORPORATE SOURCE: Pfizer Global Research and Development, Groton, CT 06340, USA.
SOURCE: DRUG AND CHEMICAL TOXICOLOGY, (2002 Aug) 25 (3) 255-66.
Journal code: 7801723. ISSN: 0148-0545.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 20020814

Last Updated on STN: 20030108
Entered Medline: 20030107

L5 ANSWER 15 OF 621 MEDLINE
TI Insulin and leptin combine additively to reduce food intake and body weight in rats.
AB Leptin and insulin are distinct adiposity signals that regulate food intake and body weight. Because recent evidence suggests that the central catabolic action of each is mediated by the hypothalamic melanocortin system, we tested the hypothesis that leptin and insulin interact within the brain, either additively or synergistically, to suppress food intake and **reduce body weight**. Using a within-subjects design, we co-administered leptin and insulin into the 3rd cerebral ventricle (i.c.v.) over a wide range of doses, and compared the combined effects to what occurred following the administration of each peptide alone. The data suggest that leptin and insulin interact sub-additively to regulate food intake and body weight over the first few hours. That is, the ability of combinations of leptin and insulin to reduce food intake and body weight was less than what would be predicted by the sum of their independent actions. Over 24 hours, however, the combined doses fit a strictly additive model. These data therefore imply a redundancy in the functional intracellular pathways or neuronal circuits that leptin and insulin utilize in the acute regulation of food intake and body weight, and they further imply that over time, the redundancy dissipates.

ACCESSION NUMBER: 2002280346 MEDLINE
DOCUMENT NUMBER: 22015606 PubMed ID: 12021212
TITLE: Insulin and leptin combine additively to reduce food intake and body weight in rats.
AUTHOR: Air Ellen L; Benoit Stephen C; Clegg Deborah J; Seeley Randy J; Woods Stephen C
CORPORATE SOURCE: Department of Biomedical Sciences, College of Medicine, University of Cincinnati, Cincinnati, Ohio 45267-0555, USA.
CONTRACT NUMBER: DK10032 (NIDDK)
DK17844 (NIDDK)
DK54080 (NIDDK)
DK54890 (NIDDK)
SOURCE: ENDOCRINOLOGY, (2002 Jun) 143 (6) 2449-52.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020522
Last Updated on STN: 20020619
Entered Medline: 20020618

L5 ANSWER 16 OF 621 MEDLINE
TI Y4 receptor knockout rescues fertility in ob/ob mice.
AB Hypothalamic neuropeptide Y (NPY) has been implicated in the regulation of energy balance and reproduction, and chronically elevated NPY levels in the hypothalamus are associated with obesity and reduced reproductive function. However, it is not known which one of the five cloned Y receptors mediates these effects. Here we show that crossing the Y4 receptor knockout mouse (Y4(-/-)) onto the ob/ob background restores the reduced plasma testosterone levels of ob/ob mice as well as the reduced testis and seminal vesicle size and morphology to control values. Fertility in the sterile ob/ob mice was greatly improved by Y4 receptor deletion, with 100% of male and 50% of female Y4(-/-),ob/ob double knockout mice producing live offspring. Development of the mammary ducts and lobuloalveoli was significantly enhanced in pregnant Y4(-/-) and Y4(-/-),ob/ob females. Consistent with the improved fertility and enhanced mammary gland development, gonadotropin releasing hormone (GnRH)

expression was significantly increased in Y4(-/-) and Y4(-/-),ob/ob animals. Y4(-/-) mice displayed lower body weight and reduced white adipose tissue mass accompanied by increased plasma levels of pancreatic polypeptide (PP). However, Y4 deficiency had no beneficial effects to **reduce body weight** or excessive adiposity of ob/ob mice. These data suggest that central Y4 receptor signaling specifically inhibits reproductive function under conditions of elevated central NPY-ergic tonus.

ACCESSION NUMBER: 2002261117 MEDLINE
DOCUMENT NUMBER: 21996060 PubMed ID: 12000791
TITLE: Y4 receptor knockout rescues fertility in ob/ob mice.
AUTHOR: Sainsbury Amanda; Schwarzer Christoph; Couzens Michelle; Jenkins Arthur; Oakes Samantha R; Ormandy Christopher J; Herzog Herbert
CORPORATE SOURCE: Neurobiology Research Program, Garvan Institute of Medical Research, St. Vincent's Hospital, Darlinghurst, Sydney NSW 2010, Australia.
SOURCE: GENES AND DEVELOPMENT, (2002 May 1) 16 (9) 1077-88. Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020510
Last Updated on STN: 20020618
Entered Medline: 20020617

L5 ANSWER 17 OF 621 MEDLINE
TI Dieting to **reduce body weight** for controlling hypertension in adults.

ACCESSION NUMBER: 2002223688 MEDLINE
DOCUMENT NUMBER: 21958179 PubMed ID: 11962043
TITLE: Dieting to **reduce body weight** for controlling hypertension in adults.
AUTHOR: Mulrow C D; Chiquette E; Angel L; Cornell J; Summerbell C; Anagnostelis B; Grimm R Jr; Brand M B
SOURCE: NURSING TIMES, (2001 May 17-23) 97 (20) 42. Journal code: 0423236. ISSN: 0954-7762.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Nursing Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020419
Last Updated on STN: 20020522
Entered Medline: 20020521

L5 ANSWER 18 OF 621 MEDLINE
TI Effects of vitamin A deficiency on the development and growth of rat embryos.
AB Effects of vitamin A deficiency on the development and growth of rat embryos were studied. Serious vitamin A deficiency markedly **reduce body weight**, body length and tail length, and induce incomplete development of skeleton and induce pathological change of brain and kidney of rat fetus. It was concluded that the development and growth of the embryos was affected and the brain and the kidney of fetus were damaged in serious vitamin A deficient rats.

ACCESSION NUMBER: 2002206041 MEDLINE
DOCUMENT NUMBER: 21937282 PubMed ID: 11938986
TITLE: Effects of vitamin A deficiency on the development and growth of rat embryos.
AUTHOR: Fan J; Zhu Q
CORPORATE SOURCE: School of Public Health, Tongji Medical University, Wuhan

	430030, China.
SOURCE:	WEI SHENG YEN CHIU [JOURNAL OF HYGIENE RESEARCH], (1999 Jul) 28 (4) 235-6. Journal code: 9426367. ISSN: 1000-8020.
PUB. COUNTRY:	China
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	Chinese
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200206
ENTRY DATE:	Entered STN: 20020410 Last Updated on STN: 20020605 Entered Medline: 20020604

L5 ANSWER 19 OF 621 MEDLINE

TI Endocrine and metabolic abnormalities involved in obesity associated with typical antipsychotic drug administration.

AB In this study, the authors assessed the endocrine system and glucose tolerance in obese and non-obese women chronically treated with typical antipsychotic drugs (AP). In particular, we tested the hypotheses that these subjects display hypogonadism and increased insulin resistance compared to healthy weight-matched controls, as these abnormalities create a tendency towards excessive body weight gain. Twenty-six AP-treated women were matched with 26 healthy women by age, body mass index and day of the menstrual cycle. The following serum variables were evaluated in each subject: glucose tolerance after an oral glucose overload, insulin, leptin, beta-endorphin, reproductive hormones, adrenal steroids and lipids. Compared to controls, AP-treated women displayed significantly higher levels of basal glucose, insulin after 60 min of the glucose overload, prolactin, thyroid stimulating hormone and beta-endorphin, with lower levels of C-Peptide, progesterone, 17-OH progesterone, androstenedione and high-density lipoprotein cholesterol. The levels of estradiol, estrone and leptin did not differ between the groups. Thus, women treated with typical AP appeared to display more insulin resistance than healthy controls, predisposing them to excessive weight gain. Insulin sensitivity might be further impaired when the subject switches to atypical AP administration. Metformin and related agents may **reduce body weight** in these subjects. The high levels of the opiate beta-endorphin suggest that opiate antagonists such as naloxone and naltrexone might be useful as well. Even though the luteal phase of the menstrual cycle appears to be severely disturbed, the normal serum levels of estradiol and estrone do not support the proposal derived from animal experimental studies about the use of estrogens or tamoxifen to counteract AP-induced obesity.

ACCESSION NUMBER:	2002049015 MEDLINE
DOCUMENT NUMBER:	21636716 PubMed ID: 11778142
TITLE:	Endocrine and metabolic abnormalities involved in obesity associated with typical antipsychotic drug administration.
AUTHOR:	Baptista T; Lacruz A; Angeles F; Silvera R; de Mendoza S; Mendoza M T; Hernandez L
CORPORATE SOURCE:	Department of Physiology, Los Andes University Medical School, Merida, Venezuela.. baptri@douglas.mcgill.ca
SOURCE:	PHARMACOPSYCHIATRY, (2001 Nov) 34 (6) 223-31. Journal code: 8402938. ISSN: 0176-3679.
PUB. COUNTRY:	Germany: Germany, Federal Republic of
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200203
ENTRY DATE:	Entered STN: 20020125 Last Updated on STN: 20020313 Entered Medline: 20020312

L5 ANSWER 20 OF 621 MEDLINE

TI Screening and treating adults for lipid disorders.

AB CONTEXT: Screening and treatment of lipid disorders in people at high risk for future coronary heart disease (CHD) events has gained wide acceptance, especially for patients with known CHD, but the proper role in people with low to medium risk is controversial. OBJECTIVE: To examine the evidence about the benefits and harms of screening and treatment of lipid disorders in adults without known cardiovascular disease for the U.S. Preventive Services Task Force. DATA SOURCES: We identified English-language articles on drug therapy, diet and exercise therapy, and screening for lipid disorders from comprehensive searches of the MEDLINE database from 1994 through July 1999. We used published systematic reviews, hand searching of relevant articles, the second Guide to Clinical Preventive Services, and extensive peer review to identify important older articles and to ensure completeness. DATA SYNTHESIS: There is strong, direct evidence that drug therapy reduces CHD events, CHD mortality, and possibly total mortality in middle-aged men (35 to 65 years) with abnormal lipids and a potential risk of CHD events greater than 1% to 2% per year. Indirect evidence suggests that drug therapy is also effective in other adults with similar levels of risk. The evidence is insufficient about benefits and harms of treating men younger than 35 years and women younger than 45 years who have abnormal lipids but no other risk factors for heart disease and low risk for CHD events (less than 1% per year). Trials of diet therapy for primary prevention have led to long-term reductions in cholesterol of 3% to 6% but have not demonstrated a reduction in CHD events overall. Exercise programs that maintain or **reduce body weight** can produce short-term reductions in total cholesterol of 3% to 6%, but longer-term results in unselected populations have found smaller or no effect. To identify accurately people with abnormal lipids, at least two measurements of total cholesterol and high-density lipoprotein cholesterol are required. The role of measuring triglycerides and the optimal screening interval are unclear from the available evidence. CONCLUSIONS: On the basis of the effectiveness of treatment, the availability of accurate and reliable tests, and the likelihood of identifying people with abnormal lipids and increased CHD risk, screening appears to be effective in middle-aged and older adults and in young adults with additional cardiovascular risk factors.

ACCESSION NUMBER: 2002008209 MEDLINE
DOCUMENT NUMBER: 21203144 PubMed ID: 11306236
TITLE: Screening and treating adults for lipid disorders.
AUTHOR: Pignone M P; Phillips C J; Atkins D; Teutsch S M; Mulrow C D; Lohr K N
CORPORATE SOURCE: Department of Medicine, School of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA.. pignone@med.unc.edu
CONTRACT NUMBER: 290-97-0011
SOURCE: AMERICAN JOURNAL OF PREVENTIVE MEDICINE, (2001 Apr) 20 (3 Suppl) 77-89.
Journal code: 8704773. ISSN: 0749-3797.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (META-ANALYSIS)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011207

L5 ANSWER 21 OF 621 MEDLINE
TI Effect of a complex tea on reducing blood lipid in rabbits.
AB In order to observe the effect of a complex tea composed of tea, ginkgo leaf, red koji and fructus lycii on reducing blood lipids in experimental rabbits, 40 rabbits weighted from 1.5 to 2.0 kg were divided randomly into 5 group: (I) normal control group, (II) hyperlipidemia model(positive control), (III) green tea control group, (IV) low dose complex tea group,

and (V) high dose complex tea group. Blood triglyceride(TG), cholesterol(TC), lower density lipoprotein(LDL) and high density lipoprotein(HDL) were measured before experiment and 6.12 weeks later. Then the pathological changes of heart, aorta and liver were observed. After 12 weeks of experiment, the results showed that, (1) hyperlipidemia characterized by high TC has been induced by high-fat feeds in group II, (2) the complex tea can **reduce body weight** and eliminate the lipids deposition in aorta and liver. (3) the complex tea can decrease serum TG, TC,LDL and increase serum HDL in the high complex tea group. The results indicate that the complex tea is more effective than green tea on reducing blood lipids in experimental rabbits.

ACCESSION NUMBER: 2002007088 MEDLINE
DOCUMENT NUMBER: 21151594 PubMed ID: 11255766
TITLE: Effect of a complex tea on reducing blood lipid in rabbits.
AUTHOR: Yan Y; Zhao X; Zhang L; Liu F
CORPORATE SOURCE: Shandong Institute of Labor Hygiene and Occupational Medicine, jinan 250062, China.
SOURCE: WEI SHENG YEN CHIU [JOURNAL OF HYGIENE RESEARCH], (2001 Jan) 30 (1) 52-4.
Journal code: 9426367. ISSN: 1000-8020.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 20020121
Last Updated on STN: 20021008
Entered Medline: 20021004

L5 ANSWER 22 OF 621 MEDLINE
TI The effects of human GH and its lipolytic fragment (AOD9604) on lipid metabolism following chronic treatment in obese mice and beta(3)-AR knock-out mice.
AB Both human GH (hGH) and a lipolytic fragment (AOD9604) synthesized from its C-terminus are capable of inducing weight loss and increasing lipolytic sensitivity following long-term treatment in mice. One mechanism by which this may occur is through an interaction with the beta-adrenergic pathway, particularly with the beta(3)-adrenergic receptors (beta(3)-AR). Here we describe how hGH and AOD9604 can **reduce body weight** and body fat in obese mice following 14 d of chronic ip administration. These results correlate with increases in the level of expression of beta(3)-AR RNA, the major lipolytic receptor found in fat cells. Importantly, both hGH and AOD9604 are capable of increasing the repressed levels of beta(3)-AR RNA in obese mice to levels comparable with those in lean mice. The importance of beta(3)-AR was verified when long-term treatment with hGH and AOD9604 in beta(3)-AR knock-out mice failed to produce the change in body weight and increase in lipolysis that was observed in wild-type control mice. However, in an acute experiment, AOD9604 was capable of increasing energy expenditure and fat oxidation in the beta(3)-AR knock-out mice. In conclusion, this study demonstrates that the lipolytic actions of both hGH and AOD9604 are not mediated directly through the beta(3)-AR although both compounds increase beta(3)-AR expression, which may subsequently contribute to enhanced lipolytic sensitivity.

ACCESSION NUMBER: 2001667472 MEDLINE
DOCUMENT NUMBER: 21569995 PubMed ID: 11713213
TITLE: The effects of human GH and its lipolytic fragment (AOD9604) on lipid metabolism following chronic treatment in obese mice and beta(3)-AR knock-out mice.
AUTHOR: Heffernan M; Summers R J; Thorburn A; Ogru E; Gianello R; Jiang W J; Ng F M
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Monash University, Clayton, Australia 3800.
SOURCE: ENDOCRINOLOGY, (2001 Dec) 142 (12) 5182-9.

Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011120
Last Updated on STN: 20020123
Entered Medline: 20011220

L5 ANSWER 23 OF 621 MEDLINE
TI Dietary medium-chain triacylglycerols suppress accumulation of body fat in a double-blind, controlled trial in healthy men and women.
AB We investigated the effect of long-term ingestion of dietary medium-chain triacylglycerols (MCT) on body weight and fat in humans. Using a double-blind, controlled protocol, we assessed the potential health benefits of MCT compared with long-chain triacylglycerols (LCT) in 78 healthy men and women [body mass index (BMI) > or = 23 kg/m(2): n = 26 (MCT), n = 30 (LCT); BMI < 23 kg/m(2): n = 15 (MCT), n = 7 (LCT)]. Changes in anthropometric variables, body weight and body fat during the 12-wk MCT treatment period were compared with those in subjects consuming the LCT diet. The subjects were asked to consume 9218 kJ/d and 60 g/d of total fat. The energy, fat, protein and carbohydrate intakes did not differ significantly between the groups. Body weight and body fat in both groups had decreased by wk 4, 8 and 12 of the study. However, in the subjects with BMI > or = 23 kg/m(2), the extent of the decrease in body weight was significantly greater in the MCT group than in the LCT group. In subjects with BMI > or = 23 kg/m(2), the loss of body fat in the MCT group (-3.86 +/- 0.3 kg) was significantly greater than that in the LCT group (-2.75 +/- 0.2 kg) at 8 wk. In addition, in subjects with BMI > or = 23 kg/m(2), the decrease in the area of subcutaneous fat in the MCT group was significantly greater than that in the LCT group at wk 4, 8 and 12. These results suggest that the MCT diet may **reduce body weight** and fat in individuals (BMI > or = 23 kg/m(2)) more than the LCT diet.
ACCESSION NUMBER: 2001641810 MEDLINE
DOCUMENT NUMBER: 21551370 PubMed ID: 11694608
TITLE: Dietary medium-chain triacylglycerols suppress accumulation of body fat in a double-blind, controlled trial in healthy men and women.
AUTHOR: Tsuji H; Kasai M; Takeuchi H; Nakamura M; Okazaki M; Kondo K
CORPORATE SOURCE: Division of Healthcare Science Research Laboratory, Nisshin Oil Mills, Ltd., Kanagawa 239-0832, Japan. Kagawa Nutrition University, Saitama 350-0288, Japan.. hiroaki.tsuji@nisshin-seiyu.co.jp
SOURCE: JOURNAL OF NUTRITION, (2001 Nov) 131 (11) 2853-9.
Journal code: 0404243. ISSN: 0022-3166.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011107
Last Updated on STN: 20020123
Entered Medline: 20011211

L5 ANSWER 24 OF 621 MEDLINE
TI Increase of fat oxidation and weight loss in obese mice caused by chronic treatment with human growth hormone or a modified C-terminal fragment.
AB OBJECTIVE: To observe the chronic effects of human growth hormone (hGH) and AOD9604 (a C-terminal fragment of hGH) on body weight, energy balance,

and substrate oxidation rates in obese (ob/ob) and lean C57BL/6Jmice. In vitro assays were used to confirm whether the effects of AOD9604 are mediated through the hGH receptor, and if this peptide is capable of cell proliferation via the hGH receptor. METHOD: Obese and lean mice were treated with hGH, AOD or saline for 14 days using mini-osmotic pumps. Body weight, caloric intake, resting energy expenditure, fat oxidation, glucose oxidation, and plasma glucose, insulin and glycerol were measured before and after treatment. BaF-BO3 cells transfected with the hGH receptor were used to measure in vitro 125I-hGH receptor binding and cell proliferation. RESULTS: Both hGH and AOD significantly reduced body weight gain in obese mice. This was associated with increased in vivo fat oxidation and increased plasma glycerol levels (an index of lipolysis). Unlike hGH, however, AOD9604 did not induce hyperglycaemia or reduce insulin secretion. AOD9604 does not compete for the hGH receptor and nor does it induce cell proliferation, unlike hGH. CONCLUSIONS: Both hGH and its C-terminal fragment **reduce body weight** gain, increase fat oxidation, and stimulate lipolysis in obese mice, yet AOD9604 does not interact with the hGH receptor. Thus, the concept of hGH behaving as a pro-hormone is further confirmed. This data shows that fragments of hGH can act in a manner novel to traditional hGH-stimulated pathways.

ACCESSION NUMBER:	2001566512 MEDLINE
DOCUMENT NUMBER:	21528792 PubMed ID: 11673763
TITLE:	Increase of fat oxidation and weight loss in obese mice caused by chronic treatment with human growth hormone or a modified C-terminal fragment.
AUTHOR:	Heffernan M A; Thorburn A W; Fam B; Summers R; Conway-Campbell B; Waters M J; Ng F M
CORPORATE SOURCE:	Department of Biochemistry and Molecular Biology, Monash University, Clayton, Victoria, Australia.
SOURCE:	INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (2001 Oct) 25 (10) 1442-9. Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY:	England: United Kingdom
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200112
ENTRY DATE:	Entered STN: 20011024 Last Updated on STN: 20020122 Entered Medline: 20011213

L5 ANSWER 25 OF 621 MEDLINE

TI Exercise as hypertension therapy.

AB In conclusion, the findings of most recent studies show that moderate-intensity aerobic exercise training can lower BP in patients with stage 1 and 2 essential hypertension. The average reduction in BP is 10.5 mm Hg for systolic and 7.6 mm Hg for diastolic BP. The reductions do not appear to be gender- or age-specific. Significant reductions in BP and LVH regression in patients with stage 3 hypertension have also been reported following aerobic exercise training. Resistance training exercise has not consistently shown to significantly lower BP and is not recommended as the only form of exercise for hypertensive patients. The exercise training program for optimal benefits should consist of 3 to 5 times per week, 30 to 60 minutes per session, at 50% to 80% of PMHR. However, exercise programs should be individualized to meet the patient's needs and abilities. Exercise intensity and duration should be manipulated to promote a safe and effective antihypertensive program. Initially, the exercise intensity should be low and the duration short. Both intensity and duration should progressive increase over a period of weeks until the desired goal, is achieved. The rate of progression must be tailored to meet individual patient needs and abilities. The exercise program for overweight or obese hypertensive patients should aim to promote a caloric expenditure of 300 to 500 Kcal per day and 1000 to 2000

Kcal per week. Such an approach, combined with a prudent diet, is likely to **reduce body weight**. The mechanisms mediating exercise-induced BP reduction are poorly understood. BP reductions appear to be independent of changes in body weight or body composition. There are also no indications of age- or gender-related differences in BP response to exercise. The use of ambulatory blood pressure measuring devices in exercise studies is not extensive. The few studies available indicate a more moderate reduction in BP than that reported by casual observations.

ACCESSION NUMBER: 2001523261 MEDLINE
DOCUMENT NUMBER: 21454712 PubMed ID: 11570120
TITLE: Exercise as hypertension therapy.
AUTHOR: Kokkinos P F; Narayan P; Papademetriou V
CORPORATE SOURCE: Department of Medicine, Veterans Affairs Medical Center, Cardiology and Hypertension Research Clinic, Washington, District of Columbia, USA.
SOURCE: CARDIOLOGY CLINICS, (2001 Aug) 19 (3) 507-16. Ref: 63
Journal code: 8300331. ISSN: 0733-8651.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20010926
Last Updated on STN: 20020205
Entered Medline: 20020204

L5 ANSWER 26 OF 621 MEDLINE
TI Nontoxic doses of suramin enhance activity of paclitaxel against lung metastases.
AB We recently reported that acidic (aFGF) and basic (bFGF) fibroblast growth factors confer a broad spectrum chemoresistance in solid tumors, and that suramin, an inhibitor of multiple growth factors including aFGF and bFGF, enhanced the in vitro antitumor activity of several anticancer drugs including paclitaxel (Song, S., et al., Proc. Natl. Acad. Sci. USA, 97: 8658-8663, 2000). The present study investigated in vitro and in vivo interaction between paclitaxel and suramin, using human PC3-LN cells which, upon i.v. injection into immunodeficient mice, yielded lung metastases in 100% of animals. In in vitro studies, conditioned medium (CM) obtained from histocultures of rat lung metastases induced a 3-fold resistance. The addition of suramin had no effect in the absence of CM but reversed the CM-induced resistance; calculations based on the IC(50) values indicate a complete reversal in the presence of <20 microM suramin. Analysis by the combination index method indicates a synergistic interaction between paclitaxel and suramin. In in vivo studies, animals with well-established lung metastases (at least five nodules of 1 mm in diameter) were treated i.v. with paclitaxel (15 mg/kg) and suramin (10 mg/kg) administered twice weekly for 3 weeks. Single-drug therapy with paclitaxel or suramin did not **reduce body weight**. Suramin alone had no antitumor activity. Paclitaxel alone reduced the tumor size by approximately 75%, reduced the density of nonapoptotic cells by approximately 70% in residual tumors, and enhanced the fraction of apoptotic cells by approximately 3-fold. The addition of suramin to paclitaxel therapy enhanced the antitumor effect, resulting in an additional 5-fold reduction of tumor size, an additional 9-fold reduction of the density of nonapoptotic cells, and an additional 30% increase in the apoptotic cell fraction. These data indicate significant enhancement of the efficacy of paclitaxel by suramin and support the use of nontoxic doses of suramin with paclitaxel in the treatment of lung cancer.

ACCESSION NUMBER: 2001462179 MEDLINE
DOCUMENT NUMBER: 21397960 PubMed ID: 11507065

TITLE: Nontoxic doses of suramin enhance activity of paclitaxel against lung metastases.
AUTHOR: Song S; Wientjes M G; Walsh C; Au J L
CORPORATE SOURCE: College of Pharmacy and James Cancer Hospital and Solove Research Institute, Ohio State University, Columbus, Ohio 43210, USA.
CONTRACT NUMBER: R01CA78577 (NCI)
R37CA49816 (NCI)
SOURCE: CANCER RESEARCH, (2001 Aug 15) 61 (16) 6145-50.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010910
Entered Medline: 20010906

L5 ANSWER 27 OF 621 MEDLINE
TI D-LEU-4]-OB3, a synthetic leptin agonist, improves hyperglycemic control in C57BL/6J ob/ob mice.
AB We have recently shown that the activity of a synthetic peptide corresponding to amino acid residues 116-130 of secreted mouse leptin is contained in a restricted sequence at the amino terminus of the peptide, between residues 116-122 (Ser-Cys-Ser-Leu-Pro-Gln-Thr, OB3). Substitution of the Leu residue at position 4 of OB3 with its D-isomer ([D-Leu-4]-OB3) enhanced the ability of OB3 (1 mg/day, ip, 7 days) to **reduce body weight** gain, food and water intake, and serum glucose in female C57BL/6J ob/ob mice. In the present study, we have utilized a pair-feeding approach to demonstrate that the antihyperglycemic action of [D-Leu-4]-OB3 is not solely due to its effects on caloric intake. One group of female C57BL/6J ob/ob mice (n=6) was fed ad libitum, and two additional groups (n=6 per group) were allowed 3.0 g food/mouse daily, an amount previously determined to satisfy [D-Leu-4]-OB3-treated mice. At the end of the 7-day test period, vehicle-injected mice fed ad libitum were approximately 10% heavier than their initial body weights, while pair-fed mice injected with vehicle and [D-Leu-4]-OB3-treated mice lost 5% of their initial body weights. After 1 day of treatment, blood glucose was reduced by 20% in pair-fed vehicle-injected mice, and by 40% in mice given [D-Leu-4]-OB3. Food restriction reduced blood glucose throughout the 7-day study, but not to levels seen in wild-type nonobese C57BL/6J mice of the same sex and age. After 2 days of treatment with [D-Leu-4]-OB3, however, blood glucose was reduced to levels comparable to those seen in wild-type nonobese mice. [D-Leu-4]-OB3 also lowered serum insulin levels by 53% when compared to mice fed ad libitum. Neither pair-feeding nor [D-Leu-4]-OB3 treatment had any apparent effect on thermogenesis. These results suggest that [D-Leu-4]-OB3 exerts its effects on serum glucose not only by suppressing caloric intake, but also through a separate effect on glucose metabolism which may involve increased tissue sensitivity to insulin.
ACCESSION NUMBER: 2001450506 MEDLINE
DOCUMENT NUMBER: 21387773 PubMed ID: 11495687
TITLE: D-LEU-4]-OB3, a synthetic leptin agonist, improves hyperglycemic control in C57BL/6J ob/ob mice.
AUTHOR: Grasso P; Rozhavskaya-Arena M; Leinung M C; Lee D W
CORPORATE SOURCE: Department of Medicine, Division of Endocrinology and Metabolism, MC-141, Albany Medical College, Albany, NY 12208, USA.. GrassoP@mail.amc.edu
SOURCE: REGULATORY PEPTIDES, (2001 Sep 15) 101 (1-3) 123-9.
Journal code: 8100479. ISSN: 0167-0115.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20020208
Entered Medline: 20020207

L5 ANSWER 28 OF 621 MEDLINE
TI Long-term (5-year) effects of a reduced-fat diet intervention in individuals with glucose intolerance.
AB OBJECTIVE: To determine whether reducing dietary fat would **reduce body weight** and improve long-term glycemia in people with glucose intolerance. RESEARCH DESIGN AND METHODS: A 5-year Follow-up of a 1-year randomized controlled trial of a reduced-fat ad libitum diet versus a usual diet. Participants with glucose intolerance (2-h blood glucose 7.0-11.0 mmol/l) were recruited from a Workforce Diabetes Survey. The group that was randomized to a reduced-fat diet participated in monthly small-group education sessions on reduced-fat eating for 1 year. Body weight and glucose tolerance were measured in 136 participants at baseline 6 months, and 1 year (end of intervention), with follow-up at 2 years (n = 104), 3 years (n = 99), and 5 years (n = 103). RESULTS: Compared with the control group, weight decreased in the reduced-fat-diet group (P < 0.0001); the greatest difference was noted at 1 year (-3.3 kg), diminished at subsequent follow-up (-3.2 kg at 2 years and -1.6 kg at 3 years), and was no longer present by 5 years (1.1 kg). Glucose tolerance also improved in patients on the reduced-fat diet; a lower proportion had type 2 diabetes or impaired glucose tolerance at 1 year (47 vs. 67%, P < 0.05), but in subsequent years, there were no differences between groups. However, the more compliant 50% of the intervention group maintained lower fasting and 2-h glucose at 5 years (P = 0.041 and P = 0.026 respectively) compared with control subjects. CONCLUSIONS: The natural history for people at high risk of developing type 2 diabetes is weight gain and deterioration in glucose tolerance. This process may be ameliorated through adherence to a reduced fat intake
ACCESSION NUMBER: 2001434779 MEDLINE
DOCUMENT NUMBER: 21212412 PubMed ID: 11315819
TITLE: Long-term (5-year) effects of a reduced-fat diet intervention in individuals with glucose intolerance.
COMMENT: Comment in: Diabetes Care. 2001 Apr;24(4):613-4
AUTHOR: Swinburn B A; Metcalf P A; Ley S J
CORPORATE SOURCE: Department of Community Health, University of Auckland, New Zealand.. swinburn@deakin.edu.au
SOURCE: DIABETES CARE, (2001 Apr) 24 (4) 619-24.
Journal code: 7805975. ISSN: 0149-5992.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010806
Last Updated on STN: 20010806
Entered Medline: 20010802

L5 ANSWER 29 OF 621 MEDLINE
TI Comparison of the metabolic effects of metformin and troglitazone on fructose-induced insulin resistance in male Sprague-Dawley rats.
AB BACKGROUND AND PURPOSE: Insulin resistance is a hallmark of the development of type 2 diabetes. Metformin and troglitazone are oral antidiabetic agents used to reduce insulin resistance. The aim of this study was to compare the metabolic effects of these two drugs in fructose-induced insulin-resistant rodents. METHODS: Male Sprague-Dawley rats were allocated to receive one of the following four treatments for 6 weeks: normal rat chow (control group, n = 7), high-fructose diet

(fructose group, n = 7), high-fructose diet plus metformin (metformin group, n = 8), or high-fructose diet plus troglitazone (troglitazone group, n = 8). Systolic blood pressure (SBP), insulin, free fatty acid (FFA), and triglyceride concentrations were measured as parameters of insulin resistance. Leptin concentration was also measured in the four groups. RESULTS: The fructose group developed significantly elevated SBP, hyperinsulinemia, and hypertriglyceridemia without significant change in body weight or leptin concentration compared with the control group. The metformin group had significantly reduced body weight (397.9 +/- 40.9 vs 470.1 +/- 59.6 g, p < 0.05), insulin concentration (14.8 +/- 10.5 vs 48.4 +/- 15.2 microU/mL, p < 0.05), triglyceride concentration (75.3 +/- 65.5 vs 250.1 +/- 95.7 mg/dL, p < 0.05), and leptin concentration (3.1 +/- 1.5 vs 6.9 +/- 2.0 ng/mL, p < 0.05) without significant change in SBP (147.8 +/- 5.8 vs 152.4 +/- 13.0 mm Hg, p > 0.05) compared with the fructose group. The troglitazone group had significantly reduced SBP (137.8 +/- 9.2 vs 152.4 +/- 13.0 mm Hg, p < 0.05), insulin concentration (15.0 +/- 13.6 vs 48.4 +/- 15.2 microU/mL, p < 0.05), FFA concentration (38.9 +/- 22.7 vs 78.7 +/- 24.6 mg/dL, p < 0.05), triglyceride concentration (67.6 +/- 32.4 vs 250.1 +/- 95.7 mg/dL, p < 0.05), and leptin concentration (4.4 +/- 2.0 vs 6.9 +/- 2.0 ng/mL, p < 0.05) without significant change in body weight (452.5 +/- 32.8 vs 470.1 +/- 59.6 g, p > 0.05) compared with the fructose group. The metabolic effects of metformin and troglitazone on insulin, FFA, triglyceride, and leptin concentrations were not significantly different. However, metformin treatment resulted in significantly lower body weight (397.9 +/- 40.9 vs 452.5 +/- 32.8 g) and troglitazone treatment in significantly lower SBP (137.8 +/- 9.2 vs 147.8 +/- 5.8 mm Hg) compared to the fructose group, after adjusting for basal values (p < 0.05). CONCLUSIONS: Both metformin and troglitazone were comparably effective in reducing insulin resistance. Metformin treatment caused body weight reduction but was not effective in reducing SBP. Troglitazone treatment lowered SBP but did not **reduce body weight**.

ACCESSION NUMBER: 2001318586 MEDLINE
DOCUMENT NUMBER: 21285507 PubMed ID: 11393112
TITLE: Comparison of the metabolic effects of metformin and troglitazone on fructose-induced insulin resistance in male Sprague-Dawley rats.
AUTHOR: Chen C C; Wang H J; Shih H C; Sheen L Y; Chang C T; Chen R H; Wang T Y
CORPORATE SOURCE: Division of Endocrinology and Metabolism, Department of Medicine, China Medical College Hospital, 2 Yuh-Der Road, Taichung, Taiwan.
SOURCE: JOURNAL OF THE FORMOSAN MEDICAL ASSOCIATION, (2001 Mar) 100 (3) 176-80.
Journal code: 9214933. ISSN: 0929-6646.
PUB. COUNTRY: China (Republic: 1949-)
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010625
Last Updated on STN: 20010625
Entered Medline: 20010621

L5 ANSWER 30 OF 621 MEDLINE
TI Influence of topiramate in the regulation of energy balance.
AB Topiramate (TPM) is a novel neurotherapeutic agent currently indicated for the treatment of epilepsy and undergoing development for other central nervous system indications including neuropathic pain, bipolar disorder, and migraine prophylaxis. TPM is synthesized from D-fructose and contains a sulfamate moiety that is essential for its pharmacologic activity. TPM has been observed to significantly **reduce body weight** in patients treated for seizure, which has prompted the realization of preclinical studies to characterize the effects of TPM in

the regulation of energy balance. Studies carried out in various strains of rats have provided good evidence for the ability of TPM to blunt energy deposition. Body composition analyses from rat trials have demonstrated that TPM inhibits fat deposition while reducing the activity of lipoprotein lipase (LPL) in various white adipose tissue depots. High doses of TPM (likely above the therapeutic dose range) have also been observed to reduce protein gain without catabolic effects. Although TPM cannot be described as a potent anorectic agent, it seems to have the ability to reduce food intake; significant reductions in food intake have been observed in female obese (fa/fa) Zucker rats and in female Wistar rats. TPM can also reduce energy deposition in the absence of alterations in food intake. This effect has been clearly emphasized in female lean (Fa/?) Zucker rats. In female Sprague-Dawley rats, TPM also increased energy expenditure and it has been observed to increase LPL activity in brown adipose tissue, which could indicate that TPM has the ability to enhance regulatory thermogenesis. In addition, TPM stimulates LPL activity in skeletal muscles, further emphasizing its potential to promote substrate oxidation. The mechanisms whereby TPM affects the regulation of energy balance have yet to be understood. TPM represents an antiepileptic drug (AED) with complex biochemical/pharmacologic actions. Its negative effects on energy deposition cannot be readily predicted from these actions, as AEDs are generally expected to stimulate body weight gain. Recent data, obtained from investigations aimed at assessing the effects of TPM on neuropeptidergic systems involved in the regulation of energy balance, have failed to demonstrate any significant effects of TPM on the neuropeptide Y and proopiomelanocortin systems. In conclusion, it is clear that TPM can reduce fat deposition by either reducing food intake or stimulating energy expenditure. The mechanisms whereby an AED such as TPM controls food intake and energy expenditure remains to be delineated.

ACCESSION NUMBER: 2001299871 MEDLINE
DOCUMENT NUMBER: 20509900 PubMed ID: 11054602
TITLE: Influence of topiramate in the regulation of energy balance.
AUTHOR: Richard D; Ferland J; Lalonde J; Samson P; Deshaies Y
CORPORATE SOURCE: Centre de recherche de l'hopital Laval et Centre de recherche sur le Metabolisme energetique de l'Universite Laval, Faculte de Medecine, Universite Laval, Quebec, Canada.. denis.richard@phs.ulaval.ca
SOURCE: NUTRITION, (2000 Oct) 16 (10) 961-6. Ref: 43
Journal code: 8802712. ISSN: 0899-9007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010604
Last Updated on STN: 20010604
Entered Medline: 20010531

L5 ANSWER 31 OF 621 MEDLINE
TI Metabolic imprinting on genetically predisposed neural circuits perpetuates obesity.
AB There is an obesity epidemic in the industrialized world that is not simply explained by excess energy intake and decreased energy expenditure. Persistent obesity develops when genetically predisposed individuals are in a chronic state of positive energy balance. Once established, the obese body weight is avidly defended against both over- and underfeeding. Animal studies have shown that lean individuals who are genetically predisposed toward obesity have abnormalities of neural function that prime them to become obese when caloric density of the diet is raised. These neural abnormalities are gradually "corrected" as obesity becomes

fully developed, suggesting that obesity is the normal state for such individuals. Thus, defense of the obese body weight may be perpetuated by the formation of new neural circuits involved in energy-homeostasis pathways that are not then easily abolished. Such neural plasticity can occur in both adult life and during nervous-system development. Early pre- and postnatal metabolic conditions (maternal diabetes, obesity, undernutrition) can lead genetically predisposed offspring to become even more obese as adults. This enhanced obesity is associated with altered brain neural circuitry, and these changes can then be passed on to subsequent generations in a feed-forward cycle of ever-increasing body weight. Thus, the metabolic perturbations associated with obesity during both brain development and adult life can produce "metabolic imprinting" on genetically predisposed neural circuits involved in energy homeostasis. Drugs that **reduce body weight** decrease the defended body weight and alter neural pathways involved in energy homeostasis but have no permanent effect on body weight or neural function in most individuals. Thus, early intervention in mothers, infants, children, and adults may be the only way to prevent the formation of permanent neural connections that promote and perpetuate obesity in genetically predisposed individuals.

ACCESSION NUMBER: 2001299865 MEDLINE
DOCUMENT NUMBER: 20509894 PubMed ID: 11054596
TITLE: Metabolic imprinting on genetically predisposed neural circuits perpetuates obesity.
AUTHOR: Levin B E
CORPORATE SOURCE: Department of Neurosciences, New Jersey Medical School, Newark, New Jersey, USA.. levin@umdnj.edu
SOURCE: NUTRITION, (2000 Oct) 16 (10) 909-15. Ref: 137
Journal code: 8802712. ISSN: 0899-9007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010604
Last Updated on STN: 20010604
Entered Medline: 20010531

L5 ANSWER 32 OF 621 MEDLINE
TI Topiramate reduces energy and fat gains in lean (Fa/?) and obese (fa/fa) Zucker rats.
AB OBJECTIVE: This study examined the effects of topiramate (TPM), a novel neurotherapeutic agent reported to **reduce body weight** in humans, on the components of energy balance in female Zucker rats. RESEARCH METHODS AND PROCEDURES: A 2 x 3 factorial experiment was performed in which two cohorts of Zucker rats differing in their phenotype (phenotype: lean, Fa/?; obese, fa/fa) were each divided into three groups defined by the dose of TPM administered (dose: TPM 0, vehicle; TPM 15, 15 mg/kg; TPM 60, 60 mg/kg). RESULTS: The reduction in body weight gain induced by TPM in both lean and obese rats reflected a decrease in total body energy gain, which was more evident in obese than in lean rats. Whereas TPM administration did not influence the intake of digestible energy in lean rats, it induced a reduction in food intake in obese animals. In lean, but not in obese rats, apparent energy expenditure (as calculated by the difference between energy intake and energy gain) was higher in rats treated with TPM than in animals administered the vehicle. The low dose of TPM decreased fat gain (with emphasis on subcutaneous fat) without affecting protein gain, whereas the high dose of the drug induced a reduction in both fat and protein gains. The effects of TPM on muscle and fat-depot weights were representative of the global effects of TPM on whole body fat and protein gains. The calculated energetic efficiency (energy gain/energy intake) was decreased

in both lean and obese rats after TPM treatment. TPM dose independently reduced hyperinsulinemia of obese rats, but it did not alter insulinemia of lean animals. DISCUSSION: The present results provide sound evidence for the ability of TPM to reduce fat and energy gains through reducing energetic efficiency in both lean and obese Zucker rats.

ACCESSION NUMBER: 2001233630 MEDLINE
DOCUMENT NUMBER: 21117357 PubMed ID: 11225714
TITLE: Topiramate reduces energy and fat gains in lean (Fa/?) and obese (fa/fa) Zucker rats.
AUTHOR: Picard F; Deshaies Y; Lalonde J; Samson P; Richard D
CORPORATE SOURCE: Centre de Recherche de l'H pital Laval, Faculte de Medecine, Universite Laval, Quebec, Canada.
SOURCE: OBESITY RESEARCH, (2000 Dec) 8 (9) 656-63.
Journal code: 9305691. ISSN: 1071-7323.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010503

L5 ANSWER 33 OF 621 MEDLINE
TI Cerulenin mimics effects of leptin on metabolic rate, food intake, and body weight independent of the melanocortin system, but unlike leptin, cerulenin fails to block neuroendocrine effects of fasting.
AB Cerulenin and a related compound, C75, have recently been reported to reduce food intake and body weight independent of leptin through a mechanism hypothesized, like leptin, to involve hypothalamic nutrition-sensitive neurons. To assess whether these inhibitors act through mechanisms similar to mechanisms engaged by leptin, ob/ob and Ay (agouti) mice, as well as fed and fasted wild-type mice, were treated with cerulenin. Like leptin, cerulenin reduced body weight and food intake and increased metabolic rate in ob/ob mice, and cerulenin produced the same effects in wild-type mice, whereas lithium chloride, at doses that produce conditioned taste aversion, reduced metabolic rate. However, in contrast to leptin, cerulenin did not prevent effects of fasting on plasma corticosterone or hypothalamic levels of neuropeptide Y, agouti-related peptide, pro-opiomelanocortin, or cocaine- and amphetamine-related peptide mRNA. Also, in contrast to leptin, cerulenin was highly effective to **reduce body weight** in Ay mice, in which obesity is caused by blockade of the melanocortin receptor. These data demonstrate that cerulenin produces metabolic effects similar to effects of leptin, but through mechanisms that are independent of, or down-stream from, both leptin and melanocortin receptors.

ACCESSION NUMBER: 2001199356 MEDLINE
DOCUMENT NUMBER: 21182669 PubMed ID: 11289036
TITLE: Cerulenin mimics effects of leptin on metabolic rate, food intake, and body weight independent of the melanocortin system, but unlike leptin, cerulenin fails to block neuroendocrine effects of fasting.
AUTHOR: Makimura H; Mizuno T M; Yang X J; Silverstein J; Beasley J; Mobbs C V
CORPORATE SOURCE: Department of Geriatrics, Fishberg Center for Neurobiology, Mount Sinai School of Medicine, New York, New York, USA.
SOURCE: DIABETES, (2001 Apr) 50 (4) 733-9.
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010425

Last Updated on STN: 20010425
Entered Medline: 20010419

L5 ANSWER 34 OF 621 MEDLINE
TI IL-12 gene therapy of leukemia with hematopoietic progenitor cells without the toxicity of systemic IL-12 treatment.
AB We have previously shown that the myeloid progenitor cell line 32Dc13 transduced with mIL-12 cDNAs (32DIL-12 cells) induces IFN-gamma and NK-cell-mediated cytotoxicity in vivo. Since systemic therapy with recombinant IL-12 protein has been shown to produce moderate to severe toxic side effects we examined whether IL-12 gene therapy with hematopoietic progenitor cells also induces systemic toxicities that are commonly associated with the administration of rIL-12 protein. Injection of large doses of IL-12 secreting 32DIL-12 cells (5 to 6 x 10(7) cells) significantly reduced mortality in mice injected with a lethal dose of 32Dp210 myeloid leukemia cells. More importantly, injection of similar doses of transduced cells failed to **reduce body weight** significantly or produce other visible signs of toxicity, i.e., fur ruffling or lethargy. There was no evidence of hematologic or hematopoietic toxicity resulting from the injection of transduced cells. In addition, microscopic examination of liver, kidney, lung, and intestine of mice injected with transduced cells revealed the absence of tissue necrosis or inflammatory response in any of these organs. Finally, 32DIL-12 cells were not found to interfere with the engraftment of syngeneic bone marrow transplant or the hematopoietic reconstitution of irradiated mice. These results demonstrate that IL-12 gene therapy with hematopoietic progenitor cells is nontoxic and provide a rationale for exploring the feasibility of treating minimal residual leukemia with IL-12 gene therapy.

ACCESSION NUMBER: 2001181570 MEDLINE
DOCUMENT NUMBER: 21112161 PubMed ID: 11161974
TITLE: IL-12 gene therapy of leukemia with hematopoietic progenitor cells without the toxicity of systemic IL-12 treatment.
AUTHOR: Xu Y X; Gao X; Janakiraman N; Chapman R A; Gautam S C
CORPORATE SOURCE: Division of Hematology/Oncology, Henry Ford Health System, Detroit, Michigan, 48202, USA.
SOURCE: CLINICAL IMMUNOLOGY, (2001 Feb) 98 (2) 180-9.
Journal code: 100883537. ISSN: 1521-6616.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

L5 ANSWER 35 OF 621 MEDLINE
TI Orlistat--a novel weight loss therapy.
AB OBJECTIVE: To review the pharmacology, pharmacokinetics, clinical safety and efficacy, drug interactions, and therapeutic issues related to the use of orlistat for treatment of obesity. DATA SOURCES: English-language articles were identified from MEDLINE (1966-July 2000), Roche Laboratories, organizational guidelines, National Institutes of Health and Food and Drug Administration Web sites, and Doctor's Guide online. Key words included obesity, orlistat, and lipase inhibitors. References were also identified from reference sections of published articles. STUDY SELECTION AND DATA EXTRACTION: Prospective, randomized, double-blind, placebo-controlled, human trials were selected for review and discussion. DATA SYNTHESIS: Orlistat is the first agent in the lipase inhibitor class of antiobesity drugs. Orlistat is minimally absorbed and has been shown to **reduce body weight** by inhibiting

absorption (by approximately 30%) of ingested dietary fat. Safety and efficacy have been established in one- and two-year double-blind, placebo-controlled trials; adverse effects were primarily, and almost exclusively, gastrointestinal. Due to its ability to block fat absorption, orlistat also has the capability to inhibit absorption of fat-soluble vitamins. Therefore, a daily multiple vitamin is recommended while taking orlistat. CONCLUSIONS: By inhibiting fat absorption, orlistat offers a new treatment modality for weight loss and maintenance. Preliminary data from clinical trials suggest that orlistat may be beneficial in patients with comorbid conditions related to obesity, such as diabetes and hyperlipidemia. However, further studies during postmarketing surveillance are needed to fully establish orlistats long-term benefits and safety. Orlistat should be considered a useful adjunctive therapy for weight loss and maintenance in obese patients (i.e., body mass index > or = 30 kg/m2 or > or = 27 kg/m2 if other risk factors are present) committed to lifestyle changes including diet, exercise, and behavioral modification.

ACCESSION NUMBER: 2001160149 MEDLINE
DOCUMENT NUMBER: 21157979 PubMed ID: 11261530
TITLE: Orlistat--a novel weight loss therapy.
AUTHOR: Lucas K H; Kaplan-Machlis B
CORPORATE SOURCE: Department of Clinical Pharmacy, West Virginia University, Charleston, USA.. klucas@hsc.wvu.edu
SOURCE: ANNALS OF PHARMACOTHERAPY, (2001 Mar) 35 (3) 314-28. Ref: 58
Journal code: 9203131. ISSN: 1060-0280.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010625
Last Updated on STN: 20010625
Entered Medline: 20010621

L5 ANSWER 36 OF 621 MEDLINE
TI Structured exercise improves physical functioning in women with stages I and II breast cancer: results of a randomized controlled trial.
AB PURPOSE: Self-directed and supervised exercise were compared with usual care in a clinical trial designed to evaluate the effect of structured exercise on physical functioning and other dimensions of health-related quality of life in women with stages I and II breast cancer. PATIENTS AND METHODS: One hundred twenty-three women with stages I and II breast cancer completed baseline evaluations of generic and disease- and site-specific health-related quality of life, aerobic capacity, and body weight. Participants were randomly allocated to one of three intervention groups: usual care (control group), self-directed exercise, or supervised exercise. Quality of life, aerobic capacity, and body weight measures were repeated at 26 weeks. The primary outcome was the change in the Short Form-36 physical functioning scale between baseline and 26 weeks. RESULTS: Physical functioning in the control group decreased by 4.1 points, whereas it increased by 5.7 points and 2.2 points in the self-directed and supervised exercise groups, respectively (P =.04). Post hoc analysis showed a moderately large (and clinically important) difference between the self-directed and control groups (9.8 points; P =.01) and a more modest difference between the supervised and control groups (6.3 points; P =.09). No significant differences between groups were observed for changes in quality of life scores. In a secondary analysis of participants stratified by type of adjuvant therapy, supervised exercise improved aerobic capacity (+3.5 mL/kg/min; P =.01) and reduced body weight (-4.8 kg; P <.05) compared with usual care only in participants not receiving chemotherapy. CONCLUSION: Physical exercise

can blunt some of the negative side effects of breast cancer treatment, including reduced physical functioning. Self-directed exercise is an effective way to improve physical functioning compared with usual care. In participants not receiving chemotherapy, supervised exercise may increase aerobic capacity and **reduce body weight** compared with usual care.

ACCESSION NUMBER: 2001148952 MEDLINE
DOCUMENT NUMBER: 21104113 PubMed ID: 11157015
TITLE: Structured exercise improves physical functioning in women with stages I and II breast cancer: results of a randomized controlled trial.
AUTHOR: Segal R; Evans W; Johnson D; Smith J; Colletta S; Gayton J; Woodard S; Wells G; Reid R
CORPORATE SOURCE: Department of Medical Oncology, Ottawa Regional Cancer Centre, Ottawa, Ontario, Canada.. roanne_segal@cancercare.on.ca
SOURCE: JOURNAL OF CLINICAL ONCOLOGY, (2001 Feb 1) 19 (3) 657-65. Journal code: 8309333. ISSN: 0732-183X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE) (RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404 Last Updated on STN: 20010404 Entered Medline: 20010315

L5 ANSWER 37 OF 621 MEDLINE
TI Effect of dietary flaxseed, flax oil and n-3 fatty acid supplement on hepatic and plasma characteristics relevant to fatty liver haemorrhagic syndrome in laying hens.
AB 1. Two experiments were carried out to investigate the effect of dietary flaxseed, flax oil and n-3 fatty acid supplementation (Dry n-3) on hepatic fat content, plasma triglycerides, hepatic haemorrhage score, egg production, food intake and body weight in an inbred line of Single Comb White Leghorns (UCD-003) predisposed to fatty liver haemorrhagic syndrome (FLHS) and normal SCWL hens. 2. Feeding diets containing 100 g/kg ground flaxseed, 40 g/kg flax oil, or 100 g/kg Dry n-3 reduced body weight and significantly reduced hepatic fat content compared to feeding the control diet with animal and vegetable oil as a fat source. 3. Hepatic malondialdehyde, an indicator of lipid peroxidation within the liver, was not significantly affected by dietary treatment. 4. Normal SCWL hens tended to have higher egg production, greater body weight, greater food intake and higher blood triglyceride concentrations than UCD-003 hens, although the strain effects were not significant. Liver weight as a percent of body weight was significantly lower in normal SCWL hens. Treatments by strain interactions were not found. 5. The result suggested that dietary flaxseed, flax oil and Dry n-3 decrease hepatic fat content and **reduce body weight**, 2 of the predisposing factors believed to contribute to FLHS onset. However, haemorrhages were still apparent in both strains regardless of treatment, indicating that other unknown underlying mechanisms may also be responsible for FLHS.

ACCESSION NUMBER: 2001128496 MEDLINE
DOCUMENT NUMBER: 21011014 PubMed ID: 11128388
TITLE: Effect of dietary flaxseed, flax oil and n-3 fatty acid supplement on hepatic and plasma characteristics relevant to fatty liver haemorrhagic syndrome in laying hens.
AUTHOR: Schuman B E; Squires E J; Leeson S
CORPORATE SOURCE: Department of Animal and Poultry Science, University of Guelph, Ontario, Canada.
SOURCE: BRITISH POULTRY SCIENCE, (2000 Sep) 41 (4) 465-72.

Journal code: 15740290R. ISSN: 0007-1668.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010301

L5 ANSWER 38 OF 621 MEDLINE

TI The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies.

AB OBJECTIVES: Low-fat high-carbohydrate diets are recommended to prevent weight gain in normal weight subjects and **reduce body weight** in overweight and obese. However, their efficacy is controversial. We evaluated the efficacy of ad libitum low-fat diets in reducing body weight in non-diabetic individuals from the results of intervention trials. DESIGN: Studies were identified from a computerized search of the Medline database from January 1966 to July 1999 and other sources. Inclusion criteria were: controlled trials lasting more than 2 months comparing ad libitum low-fat diets as the sole intervention with a control group consuming habitual diet or a medium-fat diet ad libitum. MAIN OUTCOME MEASURES: Differences in changes in dietary fat intake, energy intake and body weight. Weighted mean differences for continuous data and 95% confidence intervals (CIs) were calculated. RESULTS: Two authors independently selected the studies meeting the inclusion criteria and extracted data from 16 trials (duration of 2-12 months) with 19 intervention groups, enrolling 1910 individuals. Fourteen were randomized. Weight loss was not the primary aim in 11 studies. Before the interventions the mean proportions of dietary energy from fat in the studies were 37.7% (95% CI, 36.9-38.5) in the low-fat groups, and 37.4% (36.4-38.4) in the control groups. The low-fat intervention produced a mean fat reduction of 10.2% (8.1-12.3). Low-fat intervention groups showed a greater weight loss than control groups (3.2 kg, 95% confidence interval 1.9-4.5 kg; $P < 0.0001$), and a greater reduction in energy intake (1 138 kJ/day, 95% confidence interval 564-1712 kJ/day, $P = 0.002$). Having a body weight 10 kg higher than the average pre-treatment body weight was associated with a 2.6 +/- 0.8 kg ($P = 0.011$) greater difference in weight loss. CONCLUSION: A reduction in dietary fat without intentional restriction of energy intake causes weight loss, which is more substantial in heavier subjects.

ACCESSION NUMBER: 2001084301 MEDLINE
DOCUMENT NUMBER: 21013117 PubMed ID: 11126204
TITLE: The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies.
AUTHOR: Astrup A; Grunwald G K; Melanson E L; Saris W H; Hill J O
CORPORATE SOURCE: The Research Department of Human Nutrition and LMC, The Royal Veterinary and Agricultural University, Frederiksberg, Denmark.. ast@kvl.dk
CONTRACT NUMBER: DK42549 (NIDDK)
DK48520 (NIDDK)
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (2000 Dec) 24 (12) 1545-52.
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (META-ANALYSIS)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010118

L5 ANSWER 39 OF 621 MEDLINE

TI Disruption of feeding behavior in CRH receptor 1-deficient mice is dependent on glucocorticoids.

AB Corticotropin-releasing hormone (CRH) has been found to markedly suppress food intake and **reduce body weight**. However, it still remains to be clarified whether those effects are mediated via either the CRH receptor 1 (CRHR1) or the CRH receptor 2 (CRHR2), or both receptor subtypes. Therefore, we investigated whether CRHR1-deficient mice (CRHR1-KO) show abnormalities in body weight and feeding behavior. CRHR1-KO and wildtype mice showed no difference in the total amount of food intake. However, there was a significant disruption in the circadian distribution of food intake: CRHR1 mutants consumed significantly more food during the light period ($p<0.01$). The normal diurnal pattern could be completely restored by oral administration of corticosterone 21-sulfate (5 mg/l added to the water-based liquid diet). We therefore conclude that in CRHR1-KO mice, the disruption of feeding behavior might be causally related to glucocorticoid deficiency, but that the CRHR1 is not likely to play a critical role in the basal regulation of ingestive behavior.

ACCESSION NUMBER: 2001058129 MEDLINE
DOCUMENT NUMBER: 20339111 PubMed ID: 10884052
TITLE: Disruption of feeding behavior in CRH receptor 1-deficient mice is dependent on glucocorticoids.
AUTHOR: Muller M B; Keck M E; Zimmermann S; Holsboer F; Wurst W
CORPORATE SOURCE: Max Planck Institute of Psychiatry, Molecular Neurogenetics, Munich, Germany.
SOURCE: NEUROREPORT, (2000 Jun 26) 11 (9) 1963-6. Journal code: 9100935. ISSN: 0959-4965.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001222

L5 ANSWER 40 OF 621 MEDLINE

TI Drospirenone: pharmacology and pharmacokinetics of a unique progestogen.

AB The pharmacology and pharmacokinetics of drospirenone, a unique progestogen, are reviewed in this paper. Unlike other progestogens, drospirenone, an analogue of spironolactone, has biochemical and pharmacologic profiles similar to endogenous progesterone, especially regarding antimineralocorticoid and antiandrogenic activities. Drospirenone counteracts the estrogen-induced stimulation of the renin-angiotensin-aldosterone system and blocks testosterone from binding to androgen receptors. Because of these characteristics, it has the potential to **reduce body weight**, blood pressure, and low-density lipoprotein levels and to enhance high-density lipoprotein levels. As a combination oral contraceptive, drospirenone with ethinyl estradiol is effective and has positive effects on weight and lipid levels. Additionally, it relieves menstrually related symptoms (e.g., negative affect and water retention) that are commonly observed with other combination oral contraceptives. Based on the biochemical and pharmacodynamic data, drospirenone appears to be a viable alternative to the currently available progestogens.

ACCESSION NUMBER: 2001017689 MEDLINE
DOCUMENT NUMBER: 20480500 PubMed ID: 11024226
TITLE: Drospirenone: pharmacology and pharmacokinetics of a unique progestogen.
AUTHOR: Krattenmacher R
CORPORATE SOURCE: Berlex Laboratories, 300 Fairfield Road, 07470, Wayne, NJ, USA.. rolf_krattenmacher@berlex.com

SOURCE: CONTRACEPTION, (2000 Jul) 62 (1) 29-38. Ref: 27
Journal code: 0234361. ISSN: 0010-7824.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001109

L5 ANSWER 41 OF 621 MEDLINE
TI [Obesity: principles of drug therapy].
Adipositas: Grundlagen--medikamentose Therapie.
AB Obesity is a major global public health problem. In many instances, a combination of diet modification, increased physical activity and behavior therapy fail or are insufficient for sustained weight loss. In these situations, drug therapy may be helpful. However, drug treatment of obesity resulted in unexpected devastating events in recent years. In the late sixties, aminorex caused an epidemic of pulmonary hypertension with high mortality rates. Dexfenfluramine and phentermine were also associated with the development of pulmonary hypertension and with alarming reports of cardiac valvular abnormalities. Therefore, these drugs were withdrawn from the market. Newer drugs, like sibutramine, a serotonin and norepinephrine reuptake inhibitor, and orlistat, a specific lipase inhibitor, **reduce body weight** significantly compared to placebo. In combination with a hypocaloric diet, weight loss of three to ten kilos can be achieved. Pharmacotherapy is limited to patients with a body mass index greater than 30 kg/m2, if non-pharmacological treatment programs have failed. The drugs should be prescribed under strict medical surveillance only.
ACCESSION NUMBER: 2000476341 MEDLINE
DOCUMENT NUMBER: 20478413 PubMed ID: 11026090
TITLE: [Obesity: principles of drug therapy].
Adipositas: Grundlagen--medikamentose Therapie.
AUTHOR: Imoberdorf R; Ballmer P E
CORPORATE SOURCE: Medizinische Klinik, Kantonsspital Winterthur..
r.imoberdorf@ksw.ch
SOURCE: THERAPEUTISCHE UMSCHAU, (2000 Aug) 57 (8) 522-5. Ref: 10
Journal code: 0407224. ISSN: 0040-5930.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001113

L5 ANSWER 42 OF 621 MEDLINE
TI The future of obesity treatment.
AB Obesity is rapidly becoming a worldwide epidemic, with significant consequences in terms of clinical burden and economic costs in treating its complications, so effective new approaches are urgently needed. Development of new drugs in this therapeutic area requires a detailed understanding of the physiology underlying body weight regulation. Recently several significant advances have been made in this area, including the identification of the appetite regulating hormone, leptin, and a detailed understanding of its targets in the central nervous system (CNS), such as neuropeptide Y (NPY) and the melanocortin-4 receptor. The

observation that some humans with severe childhood-onset obesity have defects in these regulatory systems has confirmed their relevance in humans as well as in animal models, and the search is now on to produce drugs which act on these and other CNS targets such as glucagon-like peptide I and the orexins to help **reduce body weight**. Other recently identified targets outside the central nervous system include agents acting to inhibit digestive enzymes, specifically pancreatic lipase in the form of orlistat (which has recently been licensed for obesity treatment), and looking to the future, the possibility of altering energy expenditure by modulating the newly identified uncoupling proteins is being considered. It should be remembered however, that pharmacotherapy for obesity is unlikely to provide a 'magic bullet', and that diet and lifestyle changes are likely to remain the cornerstone of treatment for the foreseeable future.

ACCESSION NUMBER: 2000446660 MEDLINE
DOCUMENT NUMBER: 20451842 PubMed ID: 10997289
TITLE: The future of obesity treatment.
AUTHOR: Wilding J
CORPORATE SOURCE: University Hospital Aintree, Liverpool, UK.
SOURCE: EXS, (2000) 89 181-91. Ref: 37
Journal code: 9204529.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001024

L5 ANSWER 43 OF 621 MEDLINE
TI oCRF and CRF (6-33) depress food but not water intake in the obese Zucker rat.
AB It has previously been demonstrated that oCRF and the CRF binding protein inhibitor CRF (6-33) **reduce body-weight** gain in obese Zucker rats. We investigated whether the reduction in body-weight is attributable to altered feeding and drinking behaviour. Obese Zucker rats were fitted with osmotic mini-pumps connected to i.c.v. cannulas. Vehicle, oCRF (5 microg/day) or CRF (6-33) (25 microg/day) were infused for 7 days and the animals observed for an additional 7 days. Body-weight and food and water-intake were recorded daily at 14.00 h. In agreement with published results, oCRF and CRF (6-33) significantly reduced body-weight gain in the obese Zucker rat. In addition, food intake was reduced, whereas water consumption was unaffected.

ACCESSION NUMBER: 2000445860 MEDLINE
DOCUMENT NUMBER: 20450441 PubMed ID: 10997635
TITLE: oCRF and CRF (6-33) depress food but not water intake in the obese Zucker rat.
AUTHOR: Bjenning C A; Rimvall K
CORPORATE SOURCE: Health Care Discovery and Preclinical Development, Novo Nordisk, Malov, Denmark.. cbjg@novo.dk
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (2000 Jun) 24 Suppl 2 S140-1.
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001010

L5 ANSWER 44 OF 621 MEDLINE

TI [Diabetic somatic polyneuropathy. Pathogenesis, clinical manifestations and therapeutic concepts].
Diabetische somatische Polyneuropathie. Pathogenese, klinische Manifestationsformen und Therapie-Konzepte.

AB Diabetic polyneuropathy is the most frequent neuropathy in western countries. In Germany, there are 3.5 to 4 million diabetic patients. Diagnosis should rule out other polyneuropathies and assess two out of the five diagnostic criteria: neuropathic symptoms, neuropathic deficits, pathological nerve conduction studies, pathological quantitative sensory testing and pathological quantitative autonomic testing. So far, the pathophysiology of diabetic neuropathy remains to be fully understood. Among the various pathophysiological concepts are the Sorbitol-Myo-Inositol hypothesis attributing Myo-Inositol depletion to the accumulation of Sorbitol and Fructose, the concept of deficiency of essential fatty acids with reduced availability of gamma-linolenic-acid and prostanoids, the pseudohypoxia- and hypoxia-hypothesis attributing endothelial and axonal dysfunction and structural lesions to increased oxidative stress and free radical production. Obviously, the hyperglycemia induced generation of advanced glycation end products (AGEs) also contributes to structural dysfunctions and lesions. Elevated levels of circulating immune complexes and activated T-lymphocytes as well the identification of autoantibodies against vagus nerve or sympathetic ganglia support the concept of an immune mediated neuropathy. The reduction of neurotrophic factors such as nerve growth factor, neurotrophin-3 or insulin-like growth factors also seems to further diabetic neuropathy. The symmetrical, distally pronounced and predominantly sensory neuropathy is far more frequent than the symmetrical neuropathy with predominant motor weakness or the asymmetrical neuropathy. The painless neuropathy manifests with impaired light touch sensation, position sense, vibratory perception and diminished or absent ankle deep tendon reflexes. The painful sensory diabetic neuropathy primarily affects small nerve fibers and accounts for decreased temperature perception and paresthesias. The proximal, diabetic amyotrophy evolves subacutely or acutely, induces motor weakness of the proximal thigh and buttock muscles and is painful. Cranial nerve III-neuropathy is also painful and has an acute onset. Truncal radiculopathy follows the distribution of truncal roots and frequently causes intense pain. Autonomic neuropathy occurs with and without somatic neuropathy. The most important therapy is to attempt optimal blood glucose control, to **reduce body weight** and hyperlipidemia. Symptomatic therapy includes alpha-lipoic acid treatment, as the antioxidant seems to improve neuropathic symptoms. Aldose reductase inhibitors might reduce sorbitol and fructose production and normalize myo-inositol levels. However, there are no aldose reductase inhibitors available in Europe as yet. Evening primrose oil, containing gamma-linolenic acid, might improve nerve conduction velocities, temperature perception, muscle strength, tendon reflexes and sensory function. Substitution of nerve growth factor showed promising results in pilot studies but failed in a large-scale multicenter study. Symptomatic pain treatment can be achieved with tricyclic antidepressants, selective serotonin reuptake inhibitors, anticonvulsants such as carbamazepine, gabapentin or lamotrigine, or anti-arrhythmic drugs such as mexiletine. Topical capsaicin application should reduce neuropathic pain but also induces local discomfort in the beginning of therapy. Vasoactive substances, so far have not proven to be of major benefit in diabetic neuropathy. Physical therapy and thorough footcare are of primary importance and allow prevention of secondary complications such as foot amputations.

ACCESSION NUMBER: 2000412521 MEDLINE
DOCUMENT NUMBER: 20379542 PubMed ID: 10923253
TITLE: [Diabetic somatic polyneuropathy. Pathogenesis, clinical manifestations and therapeutic concepts].
Diabetische somatische Polyneuropathie. Pathogenese,

klinische Manifestationsformen und Therapie-Konzepte.
AUTHOR: Hilz M J; Marthol H; Neundorfer B
CORPORATE SOURCE: Neurologische Klinik mit Poliklinik, Universitat Erlangen-Nurnberg.
SOURCE: FORTSCHRITTE DER NEUROLOGIE-PSYCHIATRIE, (2000 Jun) 68 (6) 278-88. Ref: 145
Journal code: 8103137. ISSN: 0720-4299.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000907
Last Updated on STN: 20000907
Entered Medline: 20000829

L5 ANSWER 45 OF 621 MEDLINE

TI The role of dietary fat in body fatness: evidence from a preliminary meta-analysis of ad libitum low-fat dietary intervention studies.

AB The role of high-fat diets in weight gain and obesity has been questioned because of inconsistent reports in the literature concerning the efficacy of ad libitum low-fat diets to **reduce body weight**. We conducted a meta-analysis of weight loss occurring on ad libitum low-fat diets in intervention trials, and analysed the relationship between initial body weight and weight loss. We selected controlled trials lasting more than 2 months comparing ad libitum low-fat diets with a control group consuming their habitual diet or a medium-fat diet ad libitum published from 1966 to 1998. Data were included from 16 trials with a duration of 2-12 months, involving 1728 individuals. No trials on obese subjects fulfilled the inclusion criteria. The weighted difference in weight loss between intervention and control groups was 2.55 kg (95% CI, 1.5-3.5; $P < 0.0001$). Weight loss was positively and independently related to pre-treatment body weight ($r = 0.52$, $P < 0.05$) and to reduction in the percentage of energy as fat (0.37 kg/%, $P < 0.005$) in unweighted analysis. Extrapolated to a BMI of about 30 kg/m2 and assuming a 10% reduction in dietary fat, the predicted weight loss would be 4.4 kg (95% CI, 2.0 to -6.8 kg). Because weight loss was not the primary aim in 12 of the 16 studies, it is unlikely that voluntary energy restriction contributed to the weight loss. Although there is no evidence that a high intake of simple sugars contributes to passive overconsumption, carbohydrate foods with a low glycaemic index may be more satiating and exert more beneficial effects on insulin resistance and cardiovascular risk factors. Moreover, an increase in protein content up to 25% of total energy may also contribute to reducing total energy intake. In conclusion, a low-fat diet, high in protein and fibre-rich carbohydrates, mainly from different vegetables, fruits and whole grains, is highly satiating for fewer calories than fatty foods. This diet composition provides good sources of vitamins, minerals, trace elements and fibre, and may have the most beneficial effect on blood lipids and blood-pressure levels. A reduction in dietary fat without restriction of total energy intake prevents weight gain in subjects of normal weight and produces a weight loss in overweight subjects, which is highly relevant for public health.

ACCESSION NUMBER: 2000348265 MEDLINE
DOCUMENT NUMBER: 20348265 PubMed ID: 10889789
TITLE: The role of dietary fat in body fatness: evidence from a preliminary meta-analysis of ad libitum low-fat dietary intervention studies.
AUTHOR: Astrup A; Ryan L; Grunwald G K; Storgaard M; Saris W; Melanson E; Hill J O
CORPORATE SOURCE: Research Department of Human Nutrition & LMC, Royal Veterinary and Agricultural University, Frederiksberg,

Denmark.. ast@kvl.dk
CONTRACT NUMBER: DK42549 (NIDDK)
DK48520 (NIDDK)
SOURCE: BRITISH JOURNAL OF NUTRITION, (2000 Mar) 83 Suppl 1 S25-32.
Journal code: 0372547. ISSN: 0007-1145.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(META-ANALYSIS)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000728
Last Updated on STN: 20000728
Entered Medline: 20000717

L5 ANSWER 46 OF 621 MEDLINE
TI Dieting to **reduce body weight** for
controlling hypertension in adults.
AB OBJECTIVES: Evaluate whether weight-loss diets are more effective than regular diets or other antihypertensive therapies in controlling blood pressure and preventing morbidity and mortality in hypertensive adults. SEARCH STRATEGY: MEDLINE and The Cochrane Library were searched through November 1997. Trials known to experts in the field were included through June 1998. SELECTION CRITERIA: For inclusion in the review, trials were required to meet each of the following criteria: 1) randomized controlled trials with one group assigned to a weight-loss diet and the other group assigned to either normal diet or antihypertensive therapy; 2) ambulatory adults with a mean blood pressure of at least 140 mm Hg systolic and/or 90 mm Hg diastolic; 3) active intervention consisting of a calorie-restricted diet intended to produce weight loss (excluded studies simultaneously implementing multiple lifestyle interventions where the effects of weight loss could not be disaggregated); and 4) outcome measures included weight loss and blood pressure. DATA COLLECTION AND ANALYSIS: Studies were dual abstracted by two independent reviewers using a standardized form designed specifically for this review. The primary mode of analysis was qualitative; graphs of effect sizes for individual studies were also used. MAIN RESULTS: Eighteen trials were found. Only one small study of inadequate power reported morbidity and mortality outcomes. None addressed quality of life or general well being issues. In general, participants assigned to weight-reduction groups lost weight compared to control groups. Six trials involving 361 participants assessed a weight-reducing diet versus a normal diet. The data suggested weight loss in the range of 4% to 8% of body weight was associated with a decrease in blood pressure in the range of 3 mm Hg systolic and diastolic. Three trials involving 363 participants assessed a weight-reducing diet versus treatment with antihypertensive medications. These suggested that a stepped-care approach with antihypertensive medications produced greater decreases in blood pressure (in the range of 6/5 mm Hg systolic/diastolic) than did a weight-loss diet. Trials that allowed adjustment of participants' antihypertensive regimens suggested that patients required less intensive antihypertensive drug therapy if they followed a weight-reducing diet. Data was insufficient to determine the relative efficacy of weight-reduction versus changes in sodium or potassium intake or exercise. REVIEWER'S CONCLUSIONS: Weight-reducing diets in overweight hypertensive persons can affect modest weight loss in the range of 3-9% of body weight and are probably associated with modest blood pressure decreases of roughly 3 mm Hg systolic and diastolic. Weight-reducing diets may decrease dosage requirements of persons taking antihypertensive medications.
ACCESSION NUMBER: 2000257801 MEDLINE
DOCUMENT NUMBER: 20257801 PubMed ID: 10796721
TITLE: Dieting to **reduce body weight**
for controlling hypertension in adults.
AUTHOR: Mulrow C D; Chiquette E; Angel L; Cornell J; Summerbell C;

	Anagnostelis B; Grimm R Jr; Brand M B
CORPORATE SOURCE:	Audie L. Murphy Division-Ambulatory Care (11C6), 7400 Merton Minter Blvd, San Antonio, TX 78284, USA.. HTNCRG@VERDICT.UTHSCSA.EDU
SOURCE:	Cochrane Database Syst Rev, (2000) (2) CD000484. Ref: 41 Journal code: 100909747. ISSN: 1469-493X.
PUB. COUNTRY:	ENGLAND: United Kingdom
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE) General Review; (REVIEW) (REVIEW, ACADEMIC)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200007
ENTRY DATE:	Entered STN: 20000714 Last Updated on STN: 20000714 Entered Medline: 20000706

L5 ANSWER 47 OF 621 MEDLINE

TI Cholecystokinin and leptin act synergistically to **reduce body weight**.

AB Leptin, the product of the obese gene, reduces food intake and body weight in rats and mice, whereas administration of the gut-peptide CCK reduces meal size but not body weight. In the current experiments, we report that repeated daily combination of intracerebroventricular leptin and intraperitoneal CCK results in significantly greater loss of body weight than does leptin alone. However, leptin plus CCK treatment does not synergistically reduce the size of individual 30-min sucrose meals during this period, and the effect of leptin-CCK combination on daily chow intake, while significant, is small compared with the robust effects on body weight loss. This synergistic effect on body weight loss depends on a peripheral action of CCK and a central action of leptin. These data suggest a previously unsuspected role for CCK in body weight regulation that may not depend entirely on reduction of feeding behavior and suggest a strategy for enhancing the effects of leptin in leptin-resistant obese individuals.

ACCESSION NUMBER:	2000215198 MEDLINE
DOCUMENT NUMBER:	20215198 PubMed ID: 10749775
TITLE:	Cholecystokinin and leptin act synergistically to **reduce body weight**.
AUTHOR:	Matson C A; Reid D F; Cannon T A; Ritter R C
CORPORATE SOURCE:	Program for Neuroscience, Physiology, and Pharmacology, Washington State University, Pullman, Washington 99164-6520, USA.. caesia@vetmed.wsu.edu
CONTRACT NUMBER:	NS20561 (NINDS)
SOURCE:	AMERICAN JOURNAL OF PHYSIOLOGY. REGULATORY, INTEGRATIVE AND COMPARATIVE PHYSIOLOGY, (2000 Apr) 278 (4) R882-90. Journal code: 100901230. ISSN: 0363-6119.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	200005
ENTRY DATE:	Entered STN: 20000512 Last Updated on STN: 20000512 Entered Medline: 20000504

L5 ANSWER 48 OF 621 MEDLINE

TI Nutrition and obesity: prevention and treatment.

AB The increased risk of morbidity and mortality from obesity, central body fat, and weight gain, and the beneficial effects of weight reduction argue that the cost associated with obesity could be beneficially affected by prevention of weight gain or induction of weight loss. Genetic, metabolic, and demographic predictors of weight gain have been identified that allow selection of high-risk individuals. Among the metabolic

predictors are a low metabolic rate, insulin sensitivity, and a high respiratory quotient. Demographic predictors include current smokers, certain dieting behaviors, lower socio-economic class, a low level of education, use of contraceptives, status post-partum, and rapid weight gain in childhood. Several studies suggest that weight gain can be prevented. Targets for such strategies might be high-risk families, current smokers, those who are planning to stop smoking, and those with a low metabolic rate. For those who fail primary prevention, treatment may be appropriate. The greater the degree of excess weight, the greater the risk and the more appropriate treatment becomes to **reduce body weight**.

ACCESSION NUMBER: 2000180725 MEDLINE
DOCUMENT NUMBER: 20180725 PubMed ID: 10715835
TITLE: Nutrition and obesity: prevention and treatment.
AUTHOR: Bray G A
CORPORATE SOURCE: Pennington Biomedical Research Center, Baton Rouge, Louisiana, USA.
SOURCE: NUTRITION, METABOLISM, AND CARDIOVASCULAR DISEASES, (1999 Aug) 9 (4 Suppl) 21-32. Ref: 85
Journal code: 9111474. ISSN: 0939-4753.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000407
Last Updated on STN: 20000407
Entered Medline: 20000328

L5 ANSWER 49 OF 621 MEDLINE
TI Amylin: a novel action in the brain to **reduce body weight**.
AB Amylin is a 37-amino acid peptide hormone that is co-secreted with insulin by pancreatic B cells in response to a nutrient stimulus (e.g., during meals). To test the hypothesis that amylin acts within the brain to reduce long-term food intake and body weight, we examined the effects of acute and chronic 3rd-ventricular (i3vt) infusion of low doses of amylin on food intake and body weight in rats. In one experiment, separate groups of ad lib-fed male Long Evans rats were given one i3vt infusion (3 microl over 30 s) of synthetic cerebrospinal fluid vehicle or 1 to 100 pmol amylin, and food intake and body weight were monitored for 7 days. Amylin potently and dose-dependently reduced 1-h food intake, with all doses producing significant reductions. The largest dose (100 pmol) significantly reduced 24-h intake by over 30%. The effect was persistent in that both 7-day cumulative food intake and body weight change were significantly decreased over the 7 days following a single injection of 100 pmol of amylin. Other groups of rats received continuous i3vt infusion (0.5 microl/h volume) of saline or 2.0 pmol/h amylin via osmotic minipumps over 10 days. Food intake over the 10-day infusion was significantly suppressed in amylin-treated rats as compared to that of controls. Consequently, by the 4th day of infusion, amylin rats weighed significantly less than baseline relative to saline controls, and this difference persisted throughout the remainder of the infusion period. At sacrifice (Day 10), the percent of body weight from retroperitoneal fat depots was significantly lower in the amylin-treated rats, indicative of a reduction of total body adiposity. In summary, the results support the hypothesis that amylin acts as a signal to the brain contributing to the maintenance of long-term energy balance.

ACCESSION NUMBER: 2000114391 MEDLINE
DOCUMENT NUMBER: 20114391 PubMed ID: 10650969
TITLE: Amylin: a novel action in the brain to **reduce body weight**.

AUTHOR: Rushing P A; Hagan M M; Seeley R J; Lutz T A; Woods S C
CORPORATE SOURCE: Department of Psychiatry, College of Medicine, University of Cincinnati, OH 45267-0559, USA.
CONTRACT NUMBER: DK17844 (NIDDK)
DK54080 (NIDDK)
SOURCE: ENDOCRINOLOGY, (2000 Feb) 141 (2) 850-3.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210

L5 ANSWER 50 OF 621 MEDLINE
TI [Benefits and limitations of protein diets in obese patients with type 2 diabetes].
Interets et limites de la diete proteique chez le patient obese diabetique de type 2.
AB Weight excess plays a major role in the pathophysiology of type 2 diabetes but only a minority of patients succeed in following a restrictive calorie diet in the long-term, able to **reduce body weight** and maintain normoglycaemia. Very low-calorie diets such as protein diets rapidly reduce plasma glucose levels by various mechanisms, among which a significant improvement of hepatic and muscular insulin sensitivity and a partial recovery of insulin secretion. The rapidity of the hypoglycaemic action suggests that calorie restriction plays a more important role than weight loss itself. The lowering of plasma glucose levels imposes an early reduction in the doses of antidiabetic agents to avoid hypoglycaemia. Well-balanced protein diets are well tolerated, provided that they are restricted to a few weeks. The most important limitation of the protein diet is the risk of weight regain afterwards and such situation requires the maintenance of an hypocaloric diet in the long-term. Ideally, the protein diet should be integrated in a global approach including treatment of obesity, type 2 diabetes and frequently associated other risk factors.
ACCESSION NUMBER: 2000084858 MEDLINE
DOCUMENT NUMBER: 20084858 PubMed ID: 10617797
TITLE: [Benefits and limitations of protein diets in obese patients with type 2 diabetes].
Interets et limites de la diete proteique chez le patient obese diabetique de type 2.
AUTHOR: Scheen A J
CORPORATE SOURCE: Service de Diabetologie, Nutrition et Maladies metaboliques, Departement de Medecine, CHU Sart Tilman, B-4000 Liege, Belgique.
SOURCE: ANNALES D ENDOCRINOLOGIE, (1999 Dec) 60 (6) 443-50. Ref: 59
Journal code: 0116744. ISSN: 0003-4266.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210

L5 ANSWER 51 OF 621 MEDLINE
TI Proposal of a new hypothesis for the psychosomatic treatment of obesity

and its application.

AB Dieting or a change in eating habits is the most widely used approach aimed at reducing body weight. However, it is also well known that many obese people cannot **reduce body weight** substantially, no matter how hard they try, and that they soon regain whatever they do lose. The conventional approach to the treatment of obesity is to control it by prohibition or suppression of overeating, and by orders to change eating habits. This paper presented and examined a new psychosomatic approach for obesity (NPAO). Taking the story of "The North Wind and the Sun" from Aesop's Fables as a metaphor, this hypothesis is based on the reduction of overstressors through a "Sun"-type approach as opposed to a "North Wind"-type approach. This "Sun"-type approach, which incorporates 2 principles and 3 basic rules, is useful in decreasing stressors such as prohibition, suppression and orders, and increasing pleasantness, which competes with unpleasant stress. The treatment based on this hypothesis was applied to 77 subjects: 62 men (age 46.2 +/- 8.0 years) and 15 women (age 50.6 +/- 4.5 years). All subjects were given medical checks just before and 6 months after the psychosomatic approach for obesity. For a proportion of cases, maximal oxygen uptake (VO2max) was measured before and after. In the practiced group (48 cases) except for three persons who had stopped the program within 3 months after the start, body weight and body mass index fell significantly by 5.2 kg ($p < 0.001$) and 2.0 kg/m2 ($p < 0.001$) respectively, after 6 months. There were significant reductions in total cholesterol and triglyceride ($p < 0.01$, $p < 0.01$ respectively). VO2max, however, increased significantly ($p < 0.05$). The subjects' impressions of this therapy, collected after 6 months were as follows: "It was comfortable" 67.7%, "It was hard going" 8.8%, "My body has become lighter" 79.4%, "I have become more energetic" 70.5%, and "I have become happier" 64.7%. During the period of the therapy, there was no report of any appearance of new physical or mental abnormalities such as fatigue or uncomfortableness. On the other hand, there were no significant changes in any parameters except for an increase of blood sugar in the non-practiced group (26 cases). These results strongly indicate that the NPAO is easy in practice, has a high success rate, shows no rebounding, reduces body weight safely, and results in an increase of vigor.

ACCESSION NUMBER: 2000005102 MEDLINE
DOCUMENT NUMBER: 20005102 PubMed ID: 10535202
TITLE: Proposal of a new hypothesis for the psychosomatic treatment of obesity and its application.
AUTHOR: Fujino T
CORPORATE SOURCE: Institute of Health Science, Kyushu University, Kasuga.
SOURCE: FUKUOKA IGAKU ZASSHI. FUKUOKA ACTA MEDICA, (1999 Sep) 90 (9) 353-64.
Journal code: 9423321. ISSN: 0016-254X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991126

L5 ANSWER 52 OF 621 MEDLINE

TI Addition of arginine but not glycine to lysine plus methionine-enriched diets modulates serum cholesterol and liver phospholipids in rabbits.

AB Previous experiments from our laboratory showed that in rabbits fed an amino acid diet corresponding to 30% casein, enrichment of the diet with L-lysine and L-methionine caused a marked increase in serum total and LDL cholesterol levels as well as a substantial body weight loss. Both effects were partially prevented by supplementation with L-arginine. The present studies were designed to extend this earlier observation by assessing the role of different dietary amino acids in modulation of

cholesterolemic responses and body weights. In the first experiment, the original lysine and methionine-enriched diet was supplemented with glycine in an attempt to modify methionine metabolism, and thus to **reduce body weight** loss. In addition, the mechanism of action of lysine and methionine was investigated by quantitation of major liver phospholipids. The results showed that glycine addition had no effect on weight loss or hypercholesterolemia, nor did it alter plasma levels of homocyst(e)ine, an intermediate in methionine metabolism. However, enrichment of the diet with lysine and methionine (with or without glycine) significantly increased liver levels of phosphatidylcholine and the ratio of phosphatidylcholine to phosphatidylethanolamine, apparently through increased enzymatic conversion. These changes were consistent with higher lipoprotein levels and thus may explain the hypercholesterolemia. A second experiment showed that similar effects on body weights and serum cholesterol could be obtained by adding lysine and methionine to a diet containing amino acids equivalent to only 15% casein, or 15% intact casein. This approach is more physiologic and also reduces the expense of experiments designed to study the effects of lysine and methionine in more detail.

ACCESSION NUMBER: 1999429909 MEDLINE
DOCUMENT NUMBER: 99429909 PubMed ID: 10498751
TITLE: Addition of arginine but not glycine to lysine plus methionine-enriched diets modulates serum cholesterol and liver phospholipids in rabbits.
AUTHOR: Giroux I; Kurowska E M; Freeman D J; Carroll K K
CORPORATE SOURCE: Department of Biochemistry, The University of Western Ontario, London, ON, N6A 5C1, Canada.
SOURCE: JOURNAL OF NUTRITION, (1999 Oct) 129 (10) 1807-13. Journal code: 0404243. ISSN: 0022-3166.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991115

L5 ANSWER 53 OF 621 MEDLINE
TI [The role of dietary fiber and its preparations in the protection and treatment of overweight].
Znaczenie blonnika pokarmowego i jego preparatow w zapobieganiu i leczeniu nadwagi.
AB Optimal amounts of dietary fibre in the diet are regarded as a protective factor against several health disorders such as some alimentary tract diseases, atherosclerosis and coronary heart disease. It is considered that the dietary fibre may help **reduce body weight**. The preparations of dietary fibre slow gastric emptying and decrease the appetite. However, the reduction of body weight with the application of high fibre diets, but without a change in the eating habits, is not significant.

ACCESSION NUMBER: 1999319539 MEDLINE
DOCUMENT NUMBER: 99319539 PubMed ID: 10391066
TITLE: [The role of dietary fiber and its preparations in the protection and treatment of overweight].
Znaczenie blonnika pokarmowego i jego preparatow w zapobieganiu i leczeniu nadwagi.
AUTHOR: Witkowska A; Borawska M H
CORPORATE SOURCE: Samodzielnej Pracowni Bromatologii Akademii Medycznej w Bialymstoku.
SOURCE: POLSKI MERKURIUSZ LEKARSKI, (1999 Apr) 6 (34) 224-6. Ref: 24
Journal code: 9705469. ISSN: 1426-9686.
PUB. COUNTRY: Poland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: Polish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991012
Last Updated on STN: 19991012
Entered Medline: 19990930

L5 ANSWER 54 OF 621 MEDLINE
TI Randomized, double-blind trial of chitosan for body weight reduction.
AB BACKGROUND: Overweight and obesity is a prevalent and costly threat to public health. Compelling evidence links overweight and obesity with serious disorders such as cardiovascular diseases and diabetes. Dietary regimen are notoriously burdened with poor compliance. Chitosan is promoted in the US and other countries as an oral remedy to reduce fat absorption and has now been incorporated as a major constituent into several over-the-counter remedies. The primary aim of this study is to investigate the clinical effectiveness of oral chitosan for body weight reduction. METHODS: Thirty-four overweight volunteers were included in a randomized placebo-controlled double-blind trial. Subjects were assigned to receive either four capsules of chitosan or indistinguishable placebo twice daily for 28 consecutive days. Measurements were taken at baseline, after 14 and 28d of treatment. Subjects maintained their normal diet and documented the type and amount of food consumed. Adverse effects were assessed and compliance monitored. RESULTS: Data from 30 subjects were entered into an intention-to-treat analysis. After four weeks of treatment, body mass index, serum cholesterol, triglycerides, vitamin A, D, E and beta-carotene were not significantly different in subjects receiving chitosan compared to those receiving placebo. Vitamin K was significantly increased after four weeks in the chitosan group compared with placebo ($P<0.05$). Compliance was 91.5% and 96.0% for chitosan and placebo groups respectively. CONCLUSION: The above data suggest that chitosan in the administered dosage, without dietary alterations, does not **reduce body weight** in overweight subjects. No serious adverse effects were reported.
ACCESSION NUMBER: 1999296184 MEDLINE
DOCUMENT NUMBER: 99296184 PubMed ID: 10369493
TITLE: Randomized, double-blind trial of chitosan for body weight reduction.
AUTHOR: Pittler M H; Abbot N C; Harkness E F; Ernst E
CORPORATE SOURCE: Department of Complementary Medicine, School of Postgraduate Medicine and Health Sciences, University of Exeter, United Kingdom.
SOURCE: EUROPEAN JOURNAL OF CLINICAL NUTRITION, (1999 May) 53 (5) 379-81.
Journal code: 8804070. ISSN: 0954-3007.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990730
Last Updated on STN: 19990730
Entered Medline: 19990722

L5 ANSWER 55 OF 621 MEDLINE
TI Physical activity and weight maintenance.
AB Physical activity is an important component of a weight-reducing program. When combined with a low fat diet, a physical activity program can **reduce body weight** by 10-15% in individuals

complying with the program. However, even in disciplined individuals, resistance to lose fat ultimately occurs generally before the body composition status of the reduced-obese subjects is comparable to that of their lean counterparts. On the other hand, this weight loss is generally sufficient to normalize the risk profile regarding the development of diabetes and heart diseases. Therefore, this suggests that trying to lose weight beyond the threshold of spontaneous resistance to lose fat, may be unnecessary and not feasible. Furthermore, if one also considers the potential risks for health associated with large fat loss, it is probably not relevant to encourage further weight loss in reduced-obese individuals, when their metabolic profile is normalized. It is also important to emphasize that in such a context, the physical activity program must be maintained on a permanent basis to prevent body weight regain.

ACCESSION NUMBER: 1999294469 MEDLINE
DOCUMENT NUMBER: 99294469 PubMed ID: 10368003
TITLE: Physical activity and weight maintenance.
AUTHOR: Tremblay A; Doucet E; Imbeault P
CORPORATE SOURCE: Division of Kinesiology, PEPS, Laval University, Ste-Foy, Quebec, Canada.
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (1999 Apr) 23 Suppl 3 S50-4. Ref: 32
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990727
Last Updated on STN: 19990727
Entered Medline: 19990715

L5 ANSWER 56 OF 621 MEDLINE
TI Using 99mTc-DTPA radioaerosol inhalation lung scintigraphies to detect the lung injury induced by consuming Sauropus androgynus vegetable and comparison with conventional pulmonary function tests.
AB Consuming Sauropus androgynus, a Malaysian plant, to **reduce body weight** began to become fashionable in Taiwan in 1994. According to some reports, people consuming this vegetable developed lung injuries. From July to November 1995, there were 81 nonsmoking women admitted to our hospital. Thirty-six cases had respiratory symptoms/signs and the remaining 45 had no symptoms/signs. We investigated these patients with pulmonary function tests (PFT) and technetium-99m DTPA radioaerosol inhalation lung scintigraphies (DTPA lung scan), a test to evaluate the lung ventilation and alveolar epithelial permeability. Eighteen patients had abnormal results in PFT, including obstructive type (n = 17), restrictive type (n = 5), and both (n = 4). There were 33 patients with abnormalities in DTPA lung scans, including unhomogeneous deposition of DTPA radioaerosols (n = 19), faster clearance of radioaerosols from lung (n = 26), and both (n = 12). Analyzing the results, we found that the patients with respiratory symptoms had a higher incidence of abnormal results of PFT and DTPA lung scans than the patients without respiratory symptoms ($p < 0.05$). Besides, we found that the DTPA lung scan was more sensitive than chest x-ray and PFT in detecting the lung injuries related to the consumption of S. androgynus ($p < 0.05$). Consuming S. androgynus can result in symptomatic or asymptomatic lung injuries, manifested as obstructive or restrictive ventilatory impairment, unhomogeneous radioaerosol distribution, and increased alveolar epithelial permeability. In addition, measurement of the 99mTc-DTPA clearance is the most sensitive test to detect the lung injuries caused by consuming S. androgynus.

ACCESSION NUMBER: 1999141249 MEDLINE

DOCUMENT NUMBER: 99141249 PubMed ID: 9973690
TITLE: Using 99mTc-DTPA radioaerosol inhalation lung scintigraphies to detect the lung injury induced by consuming Sauropus androgynus vegetable and comparison with conventional pulmonary function tests.
AUTHOR: Kao C H; Ho Y J; Wu C L; ChangLai S P
CORPORATE SOURCE: Department of Nuclear Medicine, Taichung Veterans General Hospital, Taichung, Taiwan.. kao@vghtc.vghtc.gov.tw
SOURCE: RESPIRATION, (1999) 66 (1) 46-51.
Journal code: 0137356. ISSN: 0025-7931.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990511
Last Updated on STN: 19990511
Entered Medline: 19990429

L5 ANSWER 57 OF 621 MEDLINE
TI Health and husbandry considerations of induced molting.
AB There have been many methods proposed to induce molting. Some worked very well in practice, but others were detrimental to the health and welfare of the hens. The most effective methods use some level of feed restriction and daylength manipulation to **reduce body weight** (Hansen, 1966; Ruszler, 1974, 1984, 1996; Swanson and Bell, 1974; Brake and Carey, 1983). Weight reduction is necessary for rest and rejuvenation of body tissues. Other methods evaluated incorporated dietary imbalances using either zinc, iodine, or sodium. Pharmaceuticals have been used but have not been cost effective. In recent years there have been those who question whether molting techniques are humane. Therefore, interest has been heightened in alternate methods to induce molting. Research reported to date has been inadequate to accurately determine which methods of induced molting are the least stressful, if they in fact, cause any more stress than that experienced by the hen during a natural molt. The three or four most highly refined methods being used commercially are not generally detrimental to the health and welfare of today's laying hen, provided that they are managed in accordance with proper husbandry practices.
ACCESSION NUMBER: 1999087615 MEDLINE
DOCUMENT NUMBER: 99087615 PubMed ID: 9872580
TITLE: Health and husbandry considerations of induced molting.
AUTHOR: Ruszler P L
CORPORATE SOURCE: Department of Animal and Poultry Sciences, Virginia Polytechnic Institute and State University, Blacksburg 24061-0306, USA.. amary@vt.edu
SOURCE: POULTRY SCIENCE, (1998 Dec) 77 (12) 1789-93. Ref: 32
Journal code: 0401150. ISSN: 0032-5791.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990225

L5 ANSWER 58 OF 621 MEDLINE
TI Utility of metformin as an adjunct to hydroxycitrate/carnitine for reducing body fat in diabetics.
AB Excessive exposure of tissues to fatty acids is likely to be the chief cause of the various dysfunctions that lead to sustained hyperglycemia in

type II diabetes. These dysfunctions are likely to be substantially reversible if body fat and dietary fat can be greatly reduced. Disinhibition of hepatic fatty acid oxidation with hydroxycitrate (HCA) and carnitine has considerable potential as a new weight-loss strategy, but in diabetics runs the risk of further enhancing excessive hepatic gluconeogenesis. Since the clinical utility of metformin in diabetes is probably traceable to inhibition of gluconeogenesis, its use as an adjunct to HCA/carnitine treatment of obesity in diabetics deserves evaluation, particularly as metformin therapy itself tends to **reduce body weight**. A consideration of relevant evidence suggests that metformin therapy will not impede the activation of fatty acid oxidation by HCA/carnitine, and is likely to potentiate the appetite-suppressant and thermogenic benefits of this strategy. Indeed, since metformin has been reported to lower body weight and improve cardiovascular risk factors in obese non-diabetics, a broader application of a metformin/HCA/carnitine therapy for obesity can be contemplated.

ACCESSION NUMBER: 1999063214 MEDLINE
DOCUMENT NUMBER: 99063214 PubMed ID: 9848468
TITLE: Utility of metformin as an adjunct to hydroxycitrate/carnitine for reducing body fat in diabetics.
AUTHOR: McCarty M F
CORPORATE SOURCE: Nutrition 21, San Diego, CA 92109, USA.
SOURCE: MEDICAL HYPOTHESES, (1998 Nov) 51 (5) 399-403. Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990316
Last Updated on STN: 19990316
Entered Medline: 19990302

L5 ANSWER 59 OF 621 MEDLINE
TI SR59230A blocks beta3-adrenoceptor-linked modulation of upcoupling protein-1 and leptin in rat brown adipocytes.
AB Experimental evidence suggests that, by stimulating energy expenditure in brown fat, selective beta3-adrenoceptor agonists can **reduce body weight** in obese rodents. In order to investigate further the physiological role of beta3-adrenoceptors in brown adipocytes, we analysed the effects of selective beta3-adrenoceptor agonists and antagonists on uncoupling protein-1 and leptin gene expression in culture-differentiated brown fat cells. Our main findings were that: (i) the leptin gene is expressed in brown adipocytes; (ii) the selective beta3-adrenoceptor agonist, N[(2S)-7-carbethoxy-1,2,3,4-tetrahydronaphth-2-yl]-(2R)-2-hydroxy- 2-(3-chlorophenil)ethanamine hydrochloride (SR58611A), inhibits leptin gene while inducing uncoupling protein-1 gene expression; (iii) these opposite effects of SR58611A are antagonized by the selective beta3-adrenoceptor antagonist, SS-enantiomer 3-(2-ethylphenoxy)-1-(1S),2,3,4-tetrahydronaphth-1-ylamin ol]-(2S)-2-propanol oxalate (SR59230A), but not by the selective beta1-adrenoceptor antagonist (+/-)-[2-(3-carbamoyl-4-hydroxyphenoxy)-ethylamino]-3-[4(1-methyl-4-trifluoromethyl-2-imidazolyl)-phenoxy]-2 propanol (CGP20712A); and (iv) these effects are due to increased cyclic AMP levels. These results confirm by means of a different experimental approach that beta3-adrenoceptors play a central role in controlling the expression of genes that are important for brown fat function.

ACCESSION NUMBER: 1998382403 MEDLINE
DOCUMENT NUMBER: 98382403 PubMed ID: 9718277
TITLE: SR59230A blocks beta3-adrenoceptor-linked modulation of upcoupling protein-1 and leptin in rat brown adipocytes.
AUTHOR: Tonello C; Dioni L; Briscini L; Nisoli E; Carruba M O
CORPORATE SOURCE: Centre for Study and Research on Obesity, Department of

	Pharmacology, School of Medicine, Ospedale L. Sacco, Milan University, Italy.
SOURCE:	EUROPEAN JOURNAL OF PHARMACOLOGY, (1998 Jul 3) 352 (1) 125-9. Journal code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY:	Netherlands
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199810
ENTRY DATE:	Entered STN: 19990106 Last Updated on STN: 20000303 Entered Medline: 19981026

L5 ANSWER 60 OF 621 MEDLINE

TI A comparison of the effect of free access to reduced fat products or their full fat equivalents on food intake, body weight, blood lipids and fat-soluble antioxidants levels and haemostasis variables.

AB OBJECTIVES: To compare the effects of free access to reduced fat products or their full fat equivalents on fat and energy intake, body weight, plasma lipids and fat-soluble antioxidants concentrations and haemostasis variables. DESIGN: A multicentre open randomised controlled trial in which intervention and control groups were followed in parallel for six months. Volunteers had free access to 44 different foods either in reduced fat or full fat version, covering between 30 and 40% of energy intake. The remainder of energy intake was covered by foods bought in regular shops. SETTING: Zeist, Wageningen and Maastricht, The Netherlands. SUBJECTS: Two hundred and forty-one non-obese healthy volunteers who had no intention to lose weight. MAIN OUTCOME MEASURES: Food intake, body weight, plasma lipid, vitamin E, beta-carotene, lycopene and fibrinogen concentrations, plasma factor VII clotting activity, and plasminogen-activator-inhibitor-I antigen level. RESULTS: One hundred and three volunteers in the full fat group and 117 volunteers in the reduced fat group completed the study. Energy and fat intake from the free access products was lower in the reduced fat group, but no difference in energy and fat intake of other products occurred. Body weight, energy-, fat- and vitamin E intake and percentage of energy derived from fat decreased in the reduced fat group. No other statistical significant intervention effects were observed. Blood lipid concentrations, factor VII activity and plasminogen-inhibitor-activator-1 level were reduced after consumption of reduced fat products. CONCLUSIONS: When subjects without intention to lose weight limit fat intake by switching from ad libitum consumption of full fat products to reduced fat products body weight gain is prevented, and fat and energy intake are reduced. Such a switch may have beneficial effects on biochemical cardiovascular risk factors. We concluded that reduced fat products will help in a population strategy aimed at preventing overweight and obesity, they will also be effective in maintaining a lower body weight after slimming. Ad libitum consumption of reduced fat products will be ineffective for those individuals that want to **reduce body weight** because they are currently overweight or obese.

ACCESSION NUMBER:	1998346946 MEDLINE
DOCUMENT NUMBER:	98346946 PubMed ID: 9683389
TITLE:	A comparison of the effect of free access to reduced fat products or their full fat equivalents on food intake, body weight, blood lipids and fat-soluble antioxidants levels and haemostasis variables.
AUTHOR:	Weststrate J A; van het Hof K H; van den Berg H; Velthuis-te-Wierik E J; de Graaf C; Zimmermanns N J; Westerterp K R; Westerterp-Plantenga M S; Verboeket-van de Venne W P
CORPORATE SOURCE:	Unilever Nutrition Centre, Unilever Research Laboratorium, Vlaardingen, The Netherlands.
SOURCE:	EUROPEAN JOURNAL OF CLINICAL NUTRITION, (1998 Jun) 52 (6)

389-95.
Journal code: 8804070. ISSN: 0954-3007.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19981008
Last Updated on STN: 19981008
Entered Medline: 19980925

L5 ANSWER 61 OF 621 MEDLINE
TI Blood pressure and plasma norepinephrine responses to dexfenfluramine in obese postmenopausal women.
AB Dexfenfluramine has been shown to **reduce body weight** and lower blood pressure in obese individuals. However. it is not clear whether the blood pressure-lowering effect is due to dexfenfluramine or to the loss of weight. This project was designed to study the effect of a 5-d treatment of dexfenfluramine on blood pressure changes in obese postmenopausal women. Twenty women aged 51-60 y matched for body mass index [BMI (in kg/m2) of 34.5-50.1] were assigned to either the dexfenfluramine group (15 mg orally twice a day for 5 d) or the control group. All subjects were instructed about an isoenergetic diet. Twenty-four-hour ambulatory blood pressure, plasma catecholamines, glucose, insulin, and lipids were measured at the beginning and repeated at the conclusion of the study. On day 5 the mean systolic (SBP) and mean diastolic blood pressures (DBP) in the dexfenfluramine group were lower than those of the control group (SBP: 114+/-7 mm Hg in the dexfenfluramine group compared with 124+/-12 mm Hg in the control group, $P < 0.05$; DBP: 70+/-9 mm Hg in the dexfenfluramine group compared with 76+/-10 mm Hg in the control group, $P < 0.05$). The mean plasma norepinephrine concentration was lower in the dexfenfluramine group than in the control group (1.60+/-0.5 compared with 2.41+/-0.5 nmol/L, respectively, $P <$ 0.05). No differences were noted in epinephrine, glucose, insulin. and lipid concentrations between the two groups. We showed that a 5-d treatment of dexfenfluramine decreases blood pressure and reduces heart rate in obese postmenopausal women. Our data suggest that these effects are results of the direct action of dexfenfluramine.
ACCESSION NUMBER: 1998196957 MEDLINE
DOCUMENT NUMBER: 98196957 PubMed ID: 9537607
TITLE: Blood pressure and plasma norepinephrine responses to dexfenfluramine in obese postmenopausal women.
AUTHOR: Flechtner-Mors M; Ditschuneit H H; Yip I; Adler G
CORPORATE SOURCE: Department of Medicine, University of Ulm, Germany.. mmors@ucla.edu
SOURCE: AMERICAN JOURNAL OF CLINICAL NUTRITION, (1998 Apr) 67 (4) 611-5.
Journal code: 0376027. ISSN: 0002-9165.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199804
ENTRY DATE: Entered STN: 19980430
Last Updated on STN: 19980430
Entered Medline: 19980421

L5 ANSWER 62 OF 621 MEDLINE
TI Obesity in hypertension: effects on prognosis and treatment.
AB OBESITY AND RISK OF MORBIDITY: Obesity is becoming an increasingly

important factor in the pathogenesis of hypertension, dyslipidemia and diabetes, which together with hyperinsulinemia comprise the deadly quartet of the insulin resistance syndrome. Obesity in the absence of these other factors is only a minor risk factor, but most obesity is accompanied by one or more of these, worsening the prognosis. The presence of obesity complicates the management of hypertension, probably in large part because of the concomitant insulin resistance which adds to the pathogenetic mechanisms and subtracts from the therapeutic efficacy of many antihypertensive regimens. Unfortunately, some of the agents used to reduce obesity may further aggravate the problem through their stimulation of sympathetic nervous activity. Nonetheless, in the treatment of hypertension in most obese patients who have relatively little excess risk, attempts to **reduce body weight** should
be attempted first, through sensible dietary restrictions, increased aerobic exercise and judicious use of non-hypertensinogenic appetite suppressants. Thereby, additional motivation to lose weight may be provided by the potential of escaping or at least delaying antihypertensive drug therapy. TREATMENT OF HIGHER-RISK OBESE INDIVIDUALS: Those obese hypertensive individuals at greater risk should be immediately started on antihypertensive drug therapy along with attempts to reduce the obesity. The choice of initial and subsequent therapy should take the patient's individual needs into account. For those with dyslipidemia or diabetes, diuretics and beta-blockers should be avoided unless there are specific indications for their use (e.g. reactive sodium retention or postmyocardial infarction). In such patients, an alpha-blocker, an angiotensin converting enzyme inhibitor or a calcium antagonist may be more appropriate. If the first drug is not sufficient, combination therapy should be considered. A diuretic may be needed to overcome reactive sodium retention. Because most obese hypertensive individuals will not be able to lose much weight, effective antihypertensive drug therapy will usually be indicated.

ACCESSION NUMBER: 1998195579 MEDLINE
DOCUMENT NUMBER: 98195579 PubMed ID: 9534095
TITLE: Obesity in hypertension: effects on prognosis and treatment.
AUTHOR: Kaplan N M
CORPORATE SOURCE: University of Texas Southwestern Medical Center, Dallas 75235-8899, USA.
SOURCE: JOURNAL OF HYPERTENSION. SUPPLEMENT, (1998 Jan) 16 (1) S35-7. Ref: 25
Journal code: 8501422. ISSN: 0952-1178.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980514

L5 ANSWER 63 OF 621 MEDLINE
TI Indan analogs of fenfluramine and norfenfluramine have reduced neurotoxic potential.
AB N-Ethyl-5-trifluoromethyl-2-aminoindan (ETAI) and 5-trifluoromethyl-2-aminoindan (TAI) were synthesized to examine the effects of side-chain cyclization on the pharmacology of the anorectic drugs fenfluramine (FEN) and norfenfluramine (norFEN), respectively. ETAI and TAI inhibited synaptosomal accumulation of 5-HT but were less effective at inhibiting catecholamine uptake than FEN or norFEN, respectively. In vivo, ETAI and TAI were less neurotoxic than FEN or norFEN; decreases in the number of [3H]paroxetine-labeled 5-HT uptake sites were 50% less than the decreases produced by FEN or norFEN. Rats treated with ETAI. TAI, FEN, and norFEN

lost 10-15% of their pretreatment body weight over a 4-day period, while saline-treated control animals gained 8%. In two-lever drug discrimination (DD) assays in rats, TAI fully substituted for the 5-HT releaser/uptake inhibitor, (+)-MBDB [(+)-N-methyl-1-(1,3-benzodioxol-5-yl)-2-aminobutane]. ETAI produced only partial substitution in this test. Neither TAI nor ETAI mimicked (+)-amphetamine in the DD assay. These studies demonstrate that incorporation of the side-chain of phenylisopropylamines into the five-membered ring of a 2-aminoindan changes both the molecular pharmacology and the neurotoxic profile of FEN and norFEN, but does not diminish the drugs' ability to **reduce body weight**.

ACCESSION NUMBER: 1998171027 MEDLINE
DOCUMENT NUMBER: 98171027 PubMed ID: 9512076
TITLE: Indan analogs of fenfluramine and norfenfluramine have reduced neurotoxic potential.
AUTHOR: Cozzi N V; Frescas S; Marona-Lewicka D; Huang X; Nichols D E
CORPORATE SOURCE: Department of Medicinal Chemistry and Molecular Pharmacology, School of Pharmacy and Pharmacal Sciences, Purdue University, West Lafayette, IN 47907, USA.
CONTRACT NUMBER: DA-04758 (NIDA)
SOURCE: PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, (1998 Mar) 59 (3) 709-15.
Journal code: 0367050. ISSN: 0091-3057.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980514
Last Updated on STN: 19980514
Entered Medline: 19980505

L5 ANSWER 64 OF 621 MEDLINE
TI Final-week performance of straight-run broilers as affected by early coccidiostat withdrawal followed by increased dietary salt.
AB Three experiments were conducted to evaluate elevated dietary NaCl levels as a means of offsetting industry-observed reductions of growth, feed intake, and feed efficiency associated with early (35-d) coccidiostat withdrawal. In the first experiment, monensin (100 ppm) was withdrawn and dietary salt levels of 0.33, 0.48, 0.63, 0.78, or 0.93% provided from 35 to 42 d of age. Experiments 2 and 3 involved lasalocid (110 ppm) withdrawal and slat amounts of 0.33, 0.53, 0.73, or 0.93%. In all studies, a positive control of 0.33% salt and the coccidiostat was also given. Monensin withdrawal reduced body weight gain, which was not overcome by slat addition. Feed efficiency during the 1-wk period was improved to the level of the group receiving continued medication by salt amounts of 0.78% or above. In contrast to industry field observations, removal of lasalocid did not **reduce body weight** gain, feed intake, or water consumption, and elevation of salt levels resulted in no consistent improvements of weight gain, feed intake, or feed conversion. Water intake increased proportionally as salt concentration increased. Elevated salt levels do not appear to be a reliable means of offsetting reduced performance related to early coccidiostat withdrawal, nor were such performance problems demonstrable for lasalocid in these trials.

ACCESSION NUMBER: 1998101205 MEDLINE
DOCUMENT NUMBER: 98101205 PubMed ID: 9438275
TITLE: Final-week performance of straight-run broilers as affected by early coccidiostat withdrawal followed by increased dietary salt.
AUTHOR: Damron B L; Christmas R B
CORPORATE SOURCE: Department of Dairy and Poultry Sciences, University of Florida, Gainesville 32611-0920, USA.

SOURCE: POULTRY SCIENCE, (1997 Dec) 76 (12) 1637-40.
Journal code: 0401150. ISSN: 0032-5791.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980306
Last Updated on STN: 19980306
Entered Medline: 19980224

L5 ANSWER 65 OF 621 MEDLINE
TI Management of polycystic ovary syndrome.
AB Polycystic ovary syndrome (PCOS), also known as Stein-Leventhal Syndrome, is a condition that afflicts many women during their childbearing years. It is one of the leading causes of female infertility. Symptoms of PCOS are related to androgen excess and are not associated with estrogen deficiency. Classic symptoms include amenorrhea, hirsutism, acne, and obesity. Management of PCOS is directed by the client's concerns regarding symptoms, desire for pregnancy, and degree of morbidity related to androgen excess. First-line management of PCOS includes diet modification, weight loss, and stress management. First-line treatment for androgen excess is estrogen therapy, the combination of estrogen and progesterone being the drugs of choice. Uncomplicated amenorrhea in PCOS is managed with monthly or bimonthly administration of medroxyprogesterone. The antiestrogen clomiphene citrate has been the drug of choice for inducing ovulation. The success of any treatment plan will depend largely on the client's ability to **reduce body weight**.
ACCESSION NUMBER: 1998100324 MEDLINE
DOCUMENT NUMBER: 98100324 PubMed ID: 9437670
TITLE: Management of polycystic ovary syndrome.
AUTHOR: Marantides D
CORPORATE SOURCE: University Hospitals Health System, Cleveland, Ohio, USA.
SOURCE: NURSE PRACTITIONER, (1997 Dec) 22 (12) 34-8, 40-1. Ref: 20
Journal code: 7603663. ISSN: 0361-1817.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Nursing Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19980226
Entered Medline: 19980219

L5 ANSWER 66 OF 621 MEDLINE
TI IGF, type I IGF receptor and IGF-binding protein mRNA expression in kidney and liver of potassium-depleted and normal rats infused with IGF-I.
AB Dietary potassium (K) depletion is known to **reduce body weight** gain and organ growth, except for kidney which increases in weight. This renal hypertrophy is preceded by increased renal IGF-I levels. In the present study, we investigated IGF-I and -II, type I IGF receptor and IGF-binding protein (IGFBP) mRNA expression in liver and kidney of K-depleted and normal rats infused with vehicle or recombinant human IGF-I. Body weight gain was almost completely arrested in K-depleted rats without any stimulatory effect of IGF-I infusion. Both absolute and relative kidney weight (kidney weight/body weight) were significantly increased in K-depleted rats and this was further enhanced by IGF-I infusion. In contrast, relative liver weight was comparable in the different groups and unaffected by IGF-I infusion. IGF-I mRNA expression was significantly lower in kidney and liver of K-depleted animals whereas type I IGF receptor levels were unchanged. In contrast,

in kidney, K depletion increased IGFBP-1 and -2 mRNA expression with no additional effect of IGF-I infusion. In liver of K-depleted animals, IGFBP-1 mRNA expression was increased whereas increased IGFBP-1 and -2 mRNA expression was observed when these animals were infused with IGF-I. These observations may point towards a differential mode of action of the IGFBPs. In kidney increased IGFBP-1 and -2 mRNA expression may enhance IGF-I bioavailability with subsequent kidney growth. In liver, with clearly detectable type I IGF receptor mRNA expression, increased IGFBP levels may protect from IGF-I-induced organ growth by decreasing IGF-I bioavailability.

ACCESSION NUMBER: 97424748 MEDLINE
DOCUMENT NUMBER: 97424748 PubMed ID: 9278861
TITLE: IGF, type I IGF receptor and IGF-binding protein mRNA expression in kidney and liver of potassium-depleted and normal rats infused with IGF-I.
AUTHOR: van Neck J W; Flyvbjerg A; Schuller A G; Rosato R R; Groffen C; van Kleffens M; Lindenbergh-Kortleve D; Dorup I; Drop S L
CORPORATE SOURCE: Department of Pediatrics, Erasmus University/Sophia Children's Hospital, Rotterdam, The Netherlands.
SOURCE: JOURNAL OF MOLECULAR ENDOCRINOLOGY, (1997 Aug) 19 (1) 59-66.
Journal code: 8902617. ISSN: 0952-5041.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971024
Last Updated on STN: 20000303
Entered Medline: 19971010

L5 ANSWER 67 OF 621 MEDLINE
TI Leptin receptors in the adrenal medulla of the rat.
AB Leptin is the protein product of the recently cloned obesity gene. Leptin receptor mRNA is found in a number of central and peripheral locations. The hypothalamus is a presumed site of action. However, little is known about the specific locations of the receptor in peripheral organs. Epinephrine has potent anorectic effects and can cause weight loss by a variety of mechanisms. Excretion of epinephrine is reduced in the ob/ob mouse, which lacks leptin, suggesting an effect by leptin on the adrenal medulla. In the current study, the presence of the leptin receptor was identified on epinephrine-secreting cells in the adrenal medulla. Immunohistochemical studies found dense leptin receptor-like immunoreactivity in the adrenal medulla with no labeling in the adrenal cortex. Double immunofluorescent labeling confirmed that the leptin receptor was present on cells that were phenylethanolamine N-methyltransferase-like immunoreactive and therefore were epinephrine-secreting cells. Leptin receptor mRNA in the adrenal medulla was detected by reverse transcriptase-polymerase chain reaction, with the majority of the mRNA coding for the short isoform (Ob-Ra) of the receptor. Finally, autoradiography was performed using 125I-labeled leptin; specific binding was found in the adrenal medulla, with no specific binding in the adrenal cortex. These results suggest that leptin may have a direct effect on epinephrine-secreting cells in the adrenal medulla. Epinephrine may play a role in mediating the effects of leptin to **reduce body weight**.

ACCESSION NUMBER: 97423458 MEDLINE
DOCUMENT NUMBER: 97423458 PubMed ID: 9277400
TITLE: Leptin receptors in the adrenal medulla of the rat.
AUTHOR: Cao G Y; Considine R V; Lynn R B
CORPORATE SOURCE: Department of Medicine, Jefferson Medical College, Thomas Jefferson University, Philadelphia, Pennsylvania 19107, USA.

SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1997 Aug) 273 (2 Pt 1) E448-52.
Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971008
Last Updated on STN: 19971008
Entered Medline: 19970924

L5 ANSWER 68 OF 621 MEDLINE
TI Diet and breast cancer: studies in laboratory animals.
AB Increasing dietary fat content increases mammary gland tumorigenesis in laboratory rodents. The effect can be attributed only in part to increasing energy intake, which itself increases tumorigenesis. Restriction of dietary or energy intake, sufficient to **reduce body weight**, reduces mammary gland tumorigenesis. Consideration of these effects has led to discussion of the possible need for changes in the feeding of laboratory rodents in carcinogenesis bioassays and other chronic studies. Studies of endocrine or other growth factors for the mammary gland have not identified specific effects of dietary fat or energy. In addition, tumorigenesis in other organs responds similarly to increased fat or decreased energy intake, indicating that the mechanisms are not, or not entirely, specific for the mammary gland. Extrapolations of results between species must always be made with caution, but the marked effects of dietary fat and energy in rodent tumorigenesis models must be considered in designing diet advice for humans.
ACCESSION NUMBER: 97307054 MEDLINE
DOCUMENT NUMBER: 97307054 PubMed ID: 9164267
TITLE: Diet and breast cancer: studies in laboratory animals.
AUTHOR: Rogers A E
CORPORATE SOURCE: Boston University School of Medicine, Department of Pathology and Laboratory Medicine and Mallory Institute of Pathology, Boston, MA 02118, USA.
SOURCE: JOURNAL OF NUTRITION, (1997 May) 127 (5 Suppl) 933S-935S.
Ref: 28
Journal code: 0404243. ISSN: 0022-3166.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970630
Last Updated on STN: 19980206
Entered Medline: 19970613

L5 ANSWER 69 OF 621 MEDLINE
TI Pediatric obesity. An overview of etiology and treatment.
AB Pediatric obesity is a chronic and growing problem for which new ideas about the biologic basis of obesity offer hope for effective solutions. Prevalence of pediatric and adult obesity is increasing despite a bewildering array of treatment programs and severe psychosocial and economic costs. The definition of obesity as an increase in fat mass, not just an increase in body weight, has profound influence on the understanding and treatment of obesity. In principle, body weight is determined by a balance between energy expenditure and energy intake, but this observation does not by itself explain obesity. There is surprisingly little evidence that the obese overeat and only some evidence that the obese are more sedentary. Understanding of the biologic basis of

obesity has grown rapidly in the last few years, especially with the identification of a novel endocrine pathway involving the adipose tissue secreted hormone leptin and the leptin receptor that is expressed in the hypothalamus. Plasma leptin levels are strongly correlated with body fat mass and are regulated by feeding and fasting, insulin, glucocorticoids, and other factors, consistent with the hypothesis that leptin is involved in body weight regulation and may even be a satiety factor (Fig. 2, Table 1). Leptin injections have been shown to **reduce body weight** of primates, although human clinical trials will not be reported until summer 1997. So many peptides influencing feeding have been described that one or more may have therapeutic potential (Fig. 2, Table 1). Although the complexity of pathways regulating body weight homeostasis slowed the pace of understanding underlying mechanisms, these complexities now offer many possibilities for novel therapeutic interventions (Fig. 2). Obesity is a major risk factor for insulin resistance and diabetes, hypertension, cancer, gallbladder disease, and atherosclerosis. In particular, adults who were obese as children have increased mortality independent of adult weight. Thus, prevention programs for children and adolescents will have long-term benefits. Treatment programs focus on modification of energy intake and expenditure through decreased calorie intake and exercise programs. Behavior-modification programs have been developed to increase effectiveness of these intake and exercise programs. These programs can produce short-term weight loss. Long-term losses are more modest but achieved more successfully in children than in adults. Several drug therapies for obesity treatment recently have been approved for adults that produce sustained 5% to 10% weight losses but experience with their use in children is limited. Identification of the biochemical pathways causing obesity by genetic approaches could provide the theoretic foundation for novel, safe, and effective obesity treatments. The cloning of leptin in 1994 has already led to testing the efficacy of leptin in clinical trials that are now underway. Although novel treatments of obesity are being developed as a result of the new biology of obesity, prevention of obesity remains an important goal.

ACCESSION NUMBER: 97277540 MEDLINE
DOCUMENT NUMBER: 97277540 PubMed ID: 9130924
TITLE: Pediatric obesity. An overview of etiology and treatment.
AUTHOR: Schonfeld-Warden N; Warden C H
CORPORATE SOURCE: Department of Pediatrics, University of California, Davis, Sacramento, USA.
SOURCE: PEDIATRIC CLINICS OF NORTH AMERICA, (1997 Apr) 44 (2) 339-61. Ref: 157
Journal code: 0401126. ISSN: 0031-3955.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970523
Last Updated on STN: 19970523
Entered Medline: 19970515

L5 ANSWER 70 OF 621 MEDLINE

TI Is leptin sensitivity the link between smoking cessation and weight gain?.

AB The known association between smoking cessation and weight gain, and the suggested role of leptin in the control of body weight, led to the present study which examined the association between smoking and serum leptin concentrations. Mean serum leptin levels, independent of body mass index (BMI), were calculated in male smokers and non-smokers from Nauru, Western Samoa and Mauritius. Smokers were generally leaner than non-smokers, and of similar ages. Levels of physical activity and glucose tolerance status were similar for smokers and non-smokers in Nauru and Western Samoa, while

in Mauritius smokers were more active and less likely to be diabetic. Leptin concentrations in smokers were significantly lower than in non-smokers, even after adjusting for BMI, waist/hip ratio (WHR) or waist girth (P < or = 0.04). This association was independent of diabetes status. Smoking, via nicotinic mechanisms, may modify the sensitivity of hypothalamic leptin receptors and consequently modulate leptin synthesis and **reduce body weight**.

ACCESSION NUMBER: 97176027 MEDLINE
DOCUMENT NUMBER: 97176027 PubMed ID: 9023601
TITLE: Is leptin sensitivity the link between smoking cessation and weight gain?.
AUTHOR: Hodge A M; Westerman R A; de Courten M P; Collier G R; Zimmet P Z; Alberti K G
CORPORATE SOURCE: International Diabetes Institute, Melbourne, Australia.
CONTRACT NUMBER: DK-25446 (NIDDK)
SOURCE: INTERNATIONAL JOURNAL OF OBESITY AND RELATED METABOLIC DISORDERS, (1997 Jan) 21 (1) 50-3.
Journal code: 9313169. ISSN: 0307-0565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970630
Last Updated on STN: 20000303
Entered Medline: 19970619

L5 ANSWER 71 OF 621 MEDLINE
TI Coherent, preventive and management strategies for obesity.
AB The increased risk of morbidity and mortality from obesity, central body fat and weight gain, and the benefits of weight reduction argue that the cost associated with obesity could be beneficially affected by prevention of weight gain or induction of weight loss. Genetic, metabolic and demographic predictors of weight gain have been identified that allow the selection of high risk individuals. Among the metabolic predictors are a low metabolic rate, insulin sensitivity and a high respiratory quotient. Demographic predictors include current smokers, certain dieting behaviours, lower socioeconomic class, a low level of education, use of contraceptives, status post-partum and rapid weight gain in childhood. Several studies suggest that weight gain can be prevented. Targets for such strategies might be high risk families, current smokers, those who are planning to stop smoking and those with a low metabolic rate. For those who fail primary prevention, treatment may be appropriate. The greater the degree of excess weight, the greater the risk and the more appropriate treatment becomes to **reduce body weight**.

ACCESSION NUMBER: 97169736 MEDLINE
DOCUMENT NUMBER: 97169736 PubMed ID: 9017284
TITLE: Coherent, preventive and management strategies for obesity.
AUTHOR: Bray G A
CORPORATE SOURCE: Pennington Biomedical Research Center, Louisiana State University, Baton Rouge 70808-4124, USA.
SOURCE: CIBA FOUNDATION SYMPOSIUM, (1996) 201 228-46; discussion 246-54. Ref: 80
Journal code: 0356636. ISSN: 0300-5208.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970414
Last Updated on STN: 19970414

Entered Medline: 19970403

L5 ANSWER 72 OF 621 MEDLINE
TI Long-term octreotide treatment reduced hyperinsulinemia, excess body weight and skin lesions in severe obesity with acanthosis nigricans.
AB A boy affected by severe obesity (kg 117, Body Mass Index 37 kg/m2) and acanthosis nigricans, was treated with octreotide for 150 days (50 micrograms x three daily subcutaneous administrations). Before treatment the patient showed an exaggerated insulin (IRI) and C-peptide (CPR) response to a standard meal with a lowering in after-meal CPR/IRI molar ratio. During octreotide treatment both IRI and CPR response was reduced but CPR/IRI molar ratio rised after meal indicating an increase in hepatic insulin removal. Body weight and acanthosis nigricans were sharply reduced during treatment and the reduction was still maintained six months after the cessation of therapy. Furthermore, IRI and CPR response, as well as the behaviour of CPR/IRI molar ratio, remained within normal range. In conclusion long-term octreotide treatment has been able to correct hyperinsulinemia and to **reduce body weight** and acanthosis nigricans.

ACCESSION NUMBER: 97160187 MEDLINE
DOCUMENT NUMBER: 97160187 PubMed ID: 9007703
TITLE: Long-term octreotide treatment reduced hyperinsulinemia, excess body weight and skin lesions in severe obesity with acanthosis nigricans.
AUTHOR: Lunetta M; Di Mauro M; Le Moli R; Burrafato S
CORPORATE SOURCE: Department of Internal Medicine, University of Catania, Italy.
SOURCE: JOURNAL OF ENDOCRINOLOGICAL INVESTIGATION, (1996 Nov) 19 (10) 699-703.
Journal code: 7806594. ISSN: 0391-4097.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970424
Last Updated on STN: 19970424
Entered Medline: 19970414

L5 ANSWER 73 OF 621 MEDLINE
TI Effects of oral and intravenous rehydration on ratings of perceived exertion and thirst.
AB The purpose of this investigation was to compare the effects of oral and intravenous saline rehydration on differentiated ratings of perceived exertion (RPE) and thirst. Eight men underwent three randomly assigned rehydration treatments following a 2- to 4-h exercise-induced dehydration bout to **reduce body weight** by 4%.
Treatments included 0.45% saline infusion (i.v.), 0.45% saline oral ingestion (ORAL), and no fluid (NF). Following rehydration and rest (2 h total), subjects walked at 50% VO2max for 90 min at 36 degrees C (EX). Central RPE during ORAL was lower ($P < 0.05$) than i.v. and NF throughout EX. Local RPE during NF was higher ($P < 0.05$) than i.v. and ORAL at minutes 20 and 40 of EX and overall RPE during NF was higher ($P < 0.05$) than ORAL at minutes 20 and 40 of EX. Significant correlations were found between overall RPE and mean skin temperature for i.v. ($r = 0.72$) and NF ($r = 0.75$), and between overall RPE and thirst ratings for i.v. ($r = 0.70$). Thirst ratings were not different among trials at postdehydration. Following rehydration, thirst was higher ($P < 0.05$) during NF than i.v. and ORAL and lower ($P < 0.05$) during ORAL than i.v. at all subsequent time points. Results suggest that oral rehydration is likely to elicit lower RPE and thirst ratings compared with intravenous rehydration.

ACCESSION NUMBER: 97152581 MEDLINE
DOCUMENT NUMBER: 97152581 PubMed ID: 9000164
TITLE: Effects of oral and intravenous rehydration on ratings of

perceived exertion and thirst.

AUTHOR: Riebe D; Maresh C M; Armstrong L E; Kenefick R W; Castellani J W; Echegaray M E; Clark B A; Camaione D N

CORPORATE SOURCE: Department of Sport, Leisure and Exercise Science, University of Connecticut, Storrs 06269-1110, USA.

SOURCE: MEDICINE AND SCIENCE IN SPORTS AND EXERCISE, (1997 Jan) 29 (1) 117-24.
Journal code: 8005433. ISSN: 0195-9131.

PUB. COUNTRY: United States

DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Space Life Sciences

ENTRY MONTH: 199703

ENTRY DATE: Entered STN: 19970321
Last Updated on STN: 20000303
Entered Medline: 19970312

L5 ANSWER 74 OF 621 MEDLINE

TI Exercise as treatment for obesity.

AB In recent times, affluent societies have become less physically active, and this has undoubtedly contributed to the increased incidence of obesity. Formal programs of exercise training can **reduce body weight** and fat, but, in many cases, the changes produced by exercise are small. When combined with energy restriction, exercise results in little further weight loss, but there is a strong trend for a greater loss of body fat. Thus, during diet-induced weight loss, added exercise seems to accelerate fat loss and maintain lean body mass, a condition which may prevent a decline in RMR. It is becoming increasingly clear that weight loss is better maintained when exercise is part of a weight-reducing program. Furthermore, following a period of diet-induced weight loss, participation in regular exercise amounting to an energy expenditure of more than 1500 kcal/week will result in more successful maintenance of the lesser weight. An emphasis should be placed on adopting life-long habits conducive to weight control and overall health rather than temporary measures for weight loss. A program which encompasses regular physical activity, modest energy intake, and reduced calories from fat has the potential to meet such a goal. Regular physical activity has the potential to reverse insulin resistance, improve cardiovascular function and the blood lipid profile, and control high blood pressure. Overweight individuals can obtain these important benefits even if body weight is not completely normalized during a program of regular physical activity. This should help alleviate problems of diabetes, heart disease, and hypertension often associated with being overweight. Further research is needed to identify more specifically the optimal amount, type, and intensity of exercise needed to produce weight loss or maintain ideal body weight. To date, the best recommendation comes from the American College of Sports Medicine. Persons are urged to engage in regular physical activity which promotes a daily energy expenditure of at least 300 kcal/day and to choose from a variety of activities, in particular, those which are enjoyable and that can be continued for life.

ACCESSION NUMBER: 97131404 MEDLINE

DOCUMENT NUMBER: 97131404 PubMed ID: 8977056

TITLE: Exercise as treatment for obesity.

AUTHOR: Zachwieja J J

CORPORATE SOURCE: Exercise and Nutrition Program, Pennington Biomedical Research Center, Louisiana State University, Baton Rouge, USA.

SOURCE: ENDOCRINOLOGY AND METABOLISM CLINICS OF NORTH AMERICA, (1996 Dec) 25 (4) 965-88. Ref: 113
Journal code: 8800104. ISSN: 0889-8529.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970327
Last Updated on STN: 19970327
Entered Medline: 19970318

L5 ANSWER 75 OF 621 MEDLINE
TI Obesity treated with phototherapy: four case studies.
AB We studied the effect of phototherapy on body weight in 4 overweight women. Melatonin was measured in the serum and urine before and after 1 hr of bright light (350 cd/m2). Psychiatric self-ratings with the Comprehensive Psychopathological Rating Scale (CPRS) and Visual Analog Scale (VAS) were performed. Phototherapy (1,500 lux) was given daily at 7-9 a.m. for 10 days and thereafter twice weekly for another 4 1/2 weeks. Three of the 4 women reduced their net weight (1.5-2.4 kg) and improved in mood. All were sensitive to light. The findings indicate that phototherapy affects the melatonin-serotonin system and carbohydrate regulation and may **reduce body weight**.
ACCESSION NUMBER: 97111532 MEDLINE
DOCUMENT NUMBER: 97111532 PubMed ID: 8953334
TITLE: Obesity treated with phototherapy: four case studies.
AUTHOR: Bylesjo E I; Boman K; Wetterberg L
CORPORATE SOURCE: Department of Neurology, Umea University Hospital, Sweden.
SOURCE: INTERNATIONAL JOURNAL OF EATING DISORDERS, (1996 Dec) 20 (4) 443-46.
Journal code: 8111226. ISSN: 0276-3478.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970424
Last Updated on STN: 19970424
Entered Medline: 19970415

L5 ANSWER 76 OF 621 MEDLINE
TI Weight reduction in obese patients with rheumatoid arthritis, with preservation of body cell mass and improvement of physical fitness.
AB OBJECTIVE: To **reduce body weight** in obese patients with rheumatoid arthritis (RA) without loss of body cell mass (BCM) and without impairment of physical performance. METHODS: Nineteen overweight RA patients were studied before, during, and after a 12-week weight reducing regime consisting of reduced dietary energy intake, supplemented with a high-protein-low-energy powder preparation, and moderate physical training. Body composition was measured by a four compartment method, which by combining determinations of total body water and total body potassium allows a distinction between the two variable components of fat free mass (FFM): BCM and extracellular water (ECW). Physical fitness was measured by a bicycle exercise test. RESULTS: Mean weight loss during the study was 4.5 kg. The patients lost 9% of their initial fat mass, 3% of initial BCM and 5% of initial ECW. Physical fitness was slightly, but significantly, improved. CONCLUSION: The regime described was successful in achieving a significant weight loss with minimal loss of BCM and maintenance of physical fitness.
ACCESSION NUMBER: 96405303 MEDLINE
DOCUMENT NUMBER: 96405303 PubMed ID: 8809443
TITLE: Weight reduction in obese patients with rheumatoid arthritis, with preservation of body cell mass and improvement of physical fitness.
AUTHOR: Engelhart M; Kondrup J; Hoie L H; Andersen V; Kristensen J